EPILEPSY

AND

ITS TREATMENT

BY

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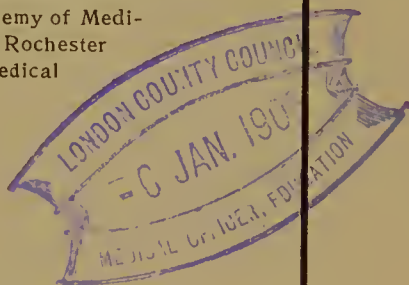Pathological Society, American Medical
Association, etc.

Fully Illustrated

DEDICATED

IN

GRATEFUL AFFECTION AND ESTEEM

TO

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OF

THE COLLEGE OF PHYSICIANS AND SURGEONS,
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MY PRECEPTOR AND FRIEND.

PREFACE.

THE great progress made in the knowledge of epilepsy and its treatment during the past decade and a half, and the fact that no complete treatise on the subject has appeared in the United States since Echeverria's work was published thirty-three years ago, were the chief reasons that led to the preparation of this volume.

In all that it treats of, including Definition, Classification, Etiology, Diagnosis, Prognosis, Pathology, Psychologic and Medicolegal Aspects, and Treatment, it is designed for the student and practitioner of medicine alike.

To present all sides of the modern care and treatment of the epileptic, I have incorporated in the chapter on general treatment notes on the education of this very generally neglected class.

The past few years have witnessed a worthy tendency to amplify the work of the physician in many cases by placing the patient under the special forms of mental, moral, physical, and hygienic *régime* now so greatly in vogue, and which are proving of such distinct value.

It is an established fact that the best results from treatment in epilepsy are obtained only when the individual, as well as his disease, is recognized and treated; and in this work it has been my aim to show how this may be most effectively accomplished.

The work represents the author's experience as Medical Superintendent of the Craig Colony for Epileptics at Sonyea, N. Y., during a period of nearly ten

years; the care and treatment of a large number of
epileptics in the New Jersey State Hospital at Morris
Plains, and his previous work in the Department of
Neurology of the Vanderbilt Clinic, Columbia College,
New York.

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The author's cordial thanks are due the publishers
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credit from the standpoint of the bookmaker's art.

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INTRODUCTION.

THE SYNONYMS OF EPILEPSY.

EPILEPSY is from the Greek word ἐπίληψις, a seizure, from ἐπιλαμβάνειν, "to seize upon."

FROM THE LATIN.

Morbus Sacer.—The sacred disease; so called because the priests of Apollo and the Sibylline priestesses were either in epileptic fits just prior to the pronouncing of prophecies, or pretended to be so.

Morbus Major.—The greater disease; referring to the severer types of epilepsy in contradistinction to the milder forms of convulsions embracing hysteria, chorea, twitching of the muscles, etc.

Morbus Herculeus.—The Herculean disease; so called possibly on account of the resistless grasp of the attack, but more probably because Hercules was a victim of it, as typified in the story of the garment of Nessus. After the destruction of Nessus, Hercules is said to have made a shirt of the hide, and having donned it could never get it off, but committed suicide to escape its torture. Some regard this story as merely a figurative description of the last stage of epilepsy, while others think it possible of better interpretation.

Morbus Commitialis.—The disease of assemblies; being so called because epileptic attacks always "had the right of way" over other matters in an assembly in ancient times, all business being suspended

until the fit was over.   Superstition and not sympathy was the governing cause of this consideration.

Morbus Mensalis.—The disease of the table; in this case the reference is doubtless due to the frequency with which convulsions were apt to appear while the patient was at the table.

Morbus Convivialis.—The disease of feasts; the meaning in this case is substantially the same as that above.

Morbus Insputatis.—The spitting disease; this may be more graphically than elegantly translated as the "slobbering sickness."

Morbus Viridellus.—The greenish disease; in all probability so called from the change an attack effects in the complexion.

Morbus Vitriolatus.—The vitriolate disease; probably an allusion to the color of ferrous sulphate, formerly called green vitriol, the temporary color of the face during an attack suggesting it.

Morbus Sonticus.—The serious or dangerous disease; in Roman law it meant *any disease excusing from duty.*   This, by the growth of the idea, gradually came to mean excusing from responsibility.

Morbus Caducus.—The falling sickness; the most commonly used of any of the ancient designations.

Morbus Unicatus.—The only disease; *unicatus* is a modern Latin word probably not existing in Latin lexicons.   Professor J. W. Lockhart, a noted Latin scholar, says: "I suppose it is used in this connection because it means there is no other disease like it, or possibly because of an imagined non-communicability of the disease."

Morbus Fœdus.—The filthy disease; so called because it produces a relaxation of the excreting orifices of the body.

Morbus Sideratus.—The star-struck disease; *sidus*, in this connection, was loosely applied to stars, planets,

and the sun. The ancients often thought the epileptic had received a blow from a star, or was blasted by the influence of some heavenly body. The frequency of the visual aura, when the patient sees flashes of light, stars, or other luminous bodies, may have helped to bestow this name.

Morbus Scelestus.—The criminal disease; alluding to the criminal tendencies it frequently develops. It appears that the term is applied to this phase of epilepsy exclusively.

Morbus Dæmoniacus.—The disease possessed by demons; a very convenient though barbarous form of ancient pathology. It usually refers to the raving phase of epilepsy.

Morbus Deificus.—The God-making disease; it seems that epilepsy acquired this name because of its potency in increasing the priest's reputation for sanctity. The symptoms of this phase were chiefly those pertaining to religious frenzy. The Greeks called it "The Priestly Disease."

Morbus Astralis.—This is only another form for the designation of the *Morbus Sideratus*, the star-struck disease previously described.

Analepsia.—Grasping upward; to seize or grasp, alluding to the victim's throwing up his hands while in a seizure and catching at nothing.

Apoplexia Parva.—Small apoplexy.

Passio Caduca et Perdito.—The falling sickness, combined with a tendency to destructiveness.

The Germans call it *epilepsie, fallsucht;* the French *epilepsie, grand mal, haut mal, petit mal;* the Italians refer to the disease as *epilepsia;* the English as *epilepsy* or *fits;* while its Scandinavian designation is *epilepsin fallendsot.*

CHAPTER I.

DEFINITION OF EPILEPSY.

It is exceedingly difficult to define a disease in which changing conditions of mind are prominent and essential features—like insanity, for instance—and it is even more difficult to define a disease that is partly mental, partly physical; more the former at one time, more the latter at another.

For this reason it is well that we are seldom called upon to define epilepsy in a single phrase or term; but the growing medicolegal importance of this disease in late years, an importance already too long neglected, requires that physicians on the witness-stand should be in a position to state in a satisfactory way how epilepsy in its great variety of forms affects the person who has it, and to tell in more or less general terms what the malady is and what it means.

Any one called upon to testify in this way would do well to make it plain that epilepsy is one of the most protean, variable, and uncertain of all maladies common to the human race, and to state that it is impossible to describe to-day how the seizures of this disease affect a given individual in a way that would make the same description applicable to-morrow, for seizure-types are not fixed in character in many cases.

If urged, however, for a description or definition, he would do well to quote from some of the standard medical writers on the subject, giving, among others, the following:

"The characteristic of the malady is the recurrence of sudden brief disturbance of some function of the

brain, varying in degree, extent, and character, but generally attended with an arrest of consciousness sufficient at least to interrupt the control of the muscles necessary for the maintenance of the erect posture."— (Sir William R. Gowers.)

"A nervous affliction characterized by sudden loss of consciousness and power of motion, with tonic and clonic convulsions, the paroxysms lasting but a short time."—(Gould.)

"Recurring attacks, sudden and very brief, of disturbance of some of the cerebral functions acting on consciousness, which are not due to a cause outside the brain."—(Thomas Clifford Allbutt.)

"Epilepsy is a chronic disease of which the characteristic symptom is a sudden trouble or loss of consciousness, this change being occasional and temporary, sometimes unattended by any evident muscular contraction, sometimes accompanied by partial spasm, and sometimes by general convulsion. The two elements probably present in every case of epilepsy are diminution of intelligence and excess of muscular contraction, and these two elements may exist in almost every degree or combination and be developed to any degree of intensity."—(Russell Reynolds.)

"Epilepsy is a disease constituted by chronic paroxysms, excited upon a direct reflex action of the medulla oblongata, in a condition of exalted irritability, coincident with sudden depression in the cerebral circulation and with the loss of consciousness, with or without muscular spasms."—(Echeverria.)

In defining epilepsy it is important to remember that it has two cardinal forms of manifestation, both of which are present in the majority of cases during the attack, but either of which may be absent, without the case losing any of its epileptic character. These manifestations are: First, impairment or loss of consciousness; second, impairment or loss of motor coördination. This being true, the following definition is perhaps as satisfactory as any that can be formulated at this time:

Epilepsy is a disease or disorder affecting the brain,

*characterized by recurrent paroxysms which are abrupt
in appearance, variable in duration but generally short,
and in which there is impairment or loss of conscious-
ness, together with impairment or loss of motor coördina-
tion, with or without convulsions.*

Should we wish to make the essential points of this
definition clearer, we might present the matter thus:

First. Impaired or lost consciousness, usually sudden
and transitory;

Second. Impaired or lost motor coördination, usually
sudden and transitory;

Third. Either condition in any case being attended
with or without convulsions.

All phenomena occurring during an epileptic attack
group themselves about the pathologic changes in
consciousness and in motility. If neither of these is
disturbed, there can be no epileptic attack.

There are epileptics who simply "forget," have
"weaknesses," "flashes," "faints," "absences,"
"blanks," and "darknesses," which may last but a few
seconds or less, or may be prolonged for days at a
time, during which the individual's physical com-
posure is in nowise disturbed. Such attacks constitute
what the author has designated as the "silent forms of
epilepsy." On the other hand, there are epileptics
who pass through the most violent and general mus-
cular contractions, during which there is enough
consciousness left for the individual to distinguish to
some extent what is going on about him and, to some
extent, remember and tell of it afterward.

Sooner or later we may come to differentiate such
convulsions as these from "true" epilepsy, calling them
epileptiform or epileptoid; for it is notable that the
minds of such patients rarely, if ever, show impairment
or disease during the interparoxysmal periods, like
the minds of those whose convulsions have a larger
element of psychic or intellectual disturbance in them.

At the same time, we must bear in mind the fact that a localized irritant, directly or reflexly affecting the motor centers, is capable of producing in time genuine convulsions in which consciousness may not always be entirely destroyed, especially during the earlier periods of the disease.

What is now called *habit epilepsy* comes under this head, meaning a type of the disease due at first to a focal irritation, central or peripheral, which being unremoved, creates a distinct pathologic basis for its existence simply through the effects of the convulsions often repeated; and so it is that simple convulsions may in time pass into genuine epilepsy, whereas primarily true epilepsy is never transformed into simple or reflex convulsions.

CHAPTER II.

CLASSIFICATION.

Based on Symptomatology: Grand Mal; Petit Mal; Psychic; Jacksonian. On Etiology (tentative and in part): Infantile Inherited; Accidental; Traumatic; Idiopathic. Similar Causes Active in Different Proportions in Early and Adult Life: Developmental; Toxic; Senile. Method of Examining for Epilepsy.

CLASSIFICATION BASED ON SYMPTOMATOLOGY.

NOTWITHSTANDING the remarkable progress that has been made during the past twelve or fifteen years in studying the etiology of epilepsy, both remote and immediate, such study has not proceeded far enough, and the time is not yet ripe for permitting a satisfactory classification based on etiology.

In order, however, to show in a measure the lines along which research is being conducted in attempting to locate causes, it seems well to present a tentative classification based on what we know about etiology at this time. This will be presented after a description has first been given of the very satisfactory classification now in use which is based wholly on symptomatology.

The latter classification is something of an anomaly in nomenclature, made up as it is of Greek, French, and English terms, requiring parts of three languages to express its four simple designations, which are as follows:

Grand Mal.
Petit Mal.
Jacksonian.
Psychic.

In *grand mal* we have a designation which describes

the severest form of epilepsy, the literal meaning of the words being great or severe illness.

By *petit mal* is meant a fit or seizure of lesser magnitude, a "little sickness," so to speak, both terms in this case being purely French.

The third designation, *Jacksonian*, is a term applied to a form of monospasm; convulsive movements confined to one arm or leg, to one side of the face, or to a single group of muscles. Attention was first called to this irregular and partial form of the disease by Hughlings-Jackson, of England, in 1866.

In the last form we employ a derivative from the Greek, ψυχικός (ψύχη, soul, mind), meaning psychic or psychical, its further meaning being "of or pertaining to the mind or soul; mental as distinguished from physical and physiologic."

Now, in order to fix this practical classification in mind, we ought to have some knowledge of the type of disease described by each variety. The following simple arrangement is offered as affording all that seems to be required:

GRAND MAL
A severe fit, the worst form of epilepsy.
One in which consciousness is always lost, and in which motor coördination is completely destroyed.
In such attacks the patient always falls or is thrown to the ground, generally with great violence.

PETIT MAL
A mild fit.
One in which consciousness need not be entirely lost; nor is motor coördination of necessity entirely destroyed.
In *petit mal* attacks the patient may not fall or be thrown to the ground, although there is some muscular involvement, either general or local.

JACKSONIAN
A form of monospasm, the convulsive movements being confined to one leg or arm, or group of muscles, and in which consciousness, as a rule, is not lost.
The patient seldom falls or is thrown to the ground, during the earlier stages of the disease, at least.
Amplification of Jacksonian attacks may in time cause complete loss of consciousness and muscular control—*grand mal*, in other words.

PSYCHIC
A seizure of the mind, leaving the body, as a rule, undisturbed.
A temporary blank in the field of consciousness.
A pathologic lapse in memory, varying from a second or two up to days and even weeks, and rarely accompanied by muscular disturbance of any kind.

Because of its simplicity and its long usage, this classification has much to commend it, especially to the student of medicine and the busy practitioner seeking to understand some of the fundamentals of the disease without being obliged to study in detail the vast array of long, tedious, scientific terms essential to a classification founded on a multitude of causes.

At the same time, this seemingly simple classification is, after all, based on a scientific footing relating to the anatomy and physiology of the brain. Thus, if a *grand mal* attack suspends or temporarily destroys all the functions of the brain, including consciousness, the special senses, and motor coördination, we can at once understand that, whatever is the lesion or process back of the fit, it affects the entire brain mass, either simultaneously or in an almost inconceivably rapid order of invasion.

If a *petit mal* attack serves only to produce slight muscular spasms, which may cause the patient to stagger but not to fall or be thrown to the ground, and some mental confusion that soon passes away without being followed by unconsciousness or coma, we can understand that the process in the brain in this case may be general in character, but not very severe; the essential difference between attacks like these and those of *grand mal* being largely one of intensity, duration, and degree. The same applies to the Jacksonian attack.

It is clear that the contraction of isolated groups of muscles, or the spasmodic movements of one leg or arm, need have but little, if any, effect on the mind, as they are due to a localized lesion in some part of the motor cortex of the brain. The psychic attack acts simply by temporarily blotting out the faculties of the mind—just that and nothing else, producing no motor disturbance whatever. It must, therefore, have its causal elements in the parts of the brain occupied

by the so-ealled *organs of the mind*, whieh the weight
of teaehing of modern physiology seems to agree in
loeating in the frontal lobes.

There is apt, as a rule, to be some confusion, some
diffieulty in differentiating attaeks of psychic epilepsy
from those of *petit mal*, but it is our belief that they
eonstitute easily distinguishable types. It will not be
out of place here to describe what these differences are,
illustrating them by referenee to two eases:

E. S., an epileptic woman of middle age, sat at the
table near my desk direeting envelopes from a printed
list of names.
I happened to be observing her at the time and
notieed that she kept writing the same name, putting
it down seven times altogether. While doing this the
only noticeable changes in her condition were a slight
drooping about the corners of the mouth, a set, staring
expression of the eyes, and some pallor of the face.
There was absolutely no impairment of the action
of any of the muscles used in writing; no sound of any
kind escaped her; no disturbance whatever of body
posture; the chief evidenee of an attaek being the
repetition in writing of the same name seven times,
which represented a blank in consciousness just long
enough to do this.
On regaining eonsciousness, whieh returned as sud-
denly and sharply as it disappeared, and as clear as it
was before the attaek, the patient was eonsiderably
embarrassed at what had oecurred, recognizing at
onee that she had repeatedly written the same name
while in a seizure. This was a typical attaek of psychie
epilepsy.
R. F. H. had set up and was regulating a camera
to get a picture of a map fastened to the wall. Two
hours before this I had observed that his faee was
slightly flushed, his eyes unusually bright, and that he
appeared animated and talkative, full of life, acting
very much like a man who had moderately indulged
in a stimulant of some kind.
He put his hand under the cloth to look at the
image on the glass, and, finding the foeus not just

right, reached out his right hand to move the screw that regulated it.

I was observing him closely, and when I saw the lens run out and back on the slideway some eight or ten times in rapid succession, doing this with his right hand, and saw him suddenly grasp one leg of the instrument with his left hand and shake it quite violently, I knew he was having a seizure. Throwing the cover off his head, I found his eyes fixed, slightly rolled upward and outward, the head turned a little to one side, the jaws going through rhythmic masticatory movements with pronounced smacking of the lips, the so-called *tasting movements* that have been referred to as constituting an aura in epilepsy.

He suddenly grew limp in the knees and acted as if he would fall to the floor, when I took hold of him and assisted him to a chair. He sat staring at me in a stupefied, dazed sort of way for possibly ten seconds, then got up and walked out of the room in a most natural way, and went over to the hospital, about two minutes' walk; he entered the dark room which he was accustomed to use for photographic work, immediately turned, came back to his camera, and, in a natural manner, continued the work of taking the picture. This was a typical convulsion of the *petit mal* type.

Both these cases are illustrations of the forms represented, but we frequently find one type taking on many of the characteristics of another, doing so to such an extent at times that type characteristics are almost completely destroyed.

More than this, some patients may have *grand mal*, *petit mal*, and psychic epilepsy all at the same time; a *grand mal* seizure may appear to-day, a *petit mal* to-morrow or the week after, while the succeeding attack may be purely psychic. This fact is especially well worth remembering when we are dealing with the medicolegal side of epilepsy. I have known numerous instances in which attacks of one kind predominated for several months or a year, to be followed

by attacks of an entirely different kind during the next year or during a number of months.

CLASSIFICATION BASED ON ETIOLOGY.

We now come to consider a classification based on etiology. To formulate even a tentative one on this basis is a scientific problem of unusual magnitude.

Many years of research in the laboratory by a class of especially trained and qualified scientists now almost unknown because of the meager opportunities offered for research in this disease, combined with additional years of studious observation of the clinical side of epilepsy and the epileptic, must intervene before we can construct an etiologic classification that will meet the scientific demand for it.

I do not, however, see any cause for being discouraged, for reflection will show that students of epilepsy are not as far behind the students of insanity in the search for causes as the casual observer might at first believe; notwithstanding the fact that insanity was a laboratory problem long before the epileptic was cared for in a way that made his scientific and clinical study in a definite manner at all possible. And then, again, epilepsy and insanity * have much in common, especially so far as psychic symptomatology goes. Studies directed to the elucidation of etiologic problems in brain pathology in insanity must also have some measure of application in the study of the epilepsies along the same line.

Let us briefly examine some points of similarity between the two diseases.

We find, in the first place, that both diseases are essentially dependent upon the brain.

In the second place, we know that continuously

* "Epilepsy and insanity," says Gowers, "certainly run together in families. The general tendency in one tends also to involve the tendency in the other, although not often in the same individual."

normal brains give off, so to speak, neither insane nor
epileptic manifestations.   These are exceedingly simple
propositions, and later on they are used expressly to
illustrate more important points.

We also know that insanity of almost every form,
whether it is melancholia, mania, paranoia, or de-
mentia, or any subdivision of these, is decidedly more
apt to be continuous in its manifestations than to
vary in any marked or radical degree.   Whatever the
cause or causes of epilepsy, the variations and irregu-
larities that spring from every form, phase, type, and
variety of the disease constitute one of its most pro-
nounced characteristics, so that in point of *constancy
of symptoms* the two diseases are widely unlike.

All these propositions will readily be granted as
true.   But let us remember that while there is a
marked variation so far as *constancy of symptoms* go,
these symptoms, when they do occur, are in many
respects identical.   The maniacal fury of the epileptic
in pre- or post-paroxysmal states, or during the so-
called psychic epileptic equivalent, does not differ
essentially in character from that of ordinary mania;
the incurable terminal dementia of the epileptic does
not differ from the terminal dementia that so fre-
quently follows all forms of mental disease.   In either
case the disintegrating and destroying action on the
brain is identical; all of which takes us back to the
original starting-point, namely, that so far as the real
basis of their manifestations go, epilepsy and insanity
have much in common; both spring from intermittent
or continuous morbid cerebral states or disease—
neither is possible in any other way.

In searching for a guide in formulating an etiologic
classification, we are impressed with the apparent
confusion that marks the incursions of scientific re-
search into this scantily explored field of medicine at
the present time.

There is a notable lack of attempt at uniformity in nomenclature on the part of such eminent authors as Hack Tuke, Gowers, Féré, Binswanger, Nothnagel, Voisin, Hughlings-Jackson, and others; and it is of interest to present the views of some of these to show how perplexing and mystifying the subject is to the student who wants first of all to grasp a few elemental facts—to get hold of a framework of established truths about which he can build as simply or as elaborately as he may choose.

From among the thirty-odd designations of epilepsy by Tuke in his most admirable "Dictionary of Psychological Medicine,"* we select the following:

Abortive Epilepsy.—A seizure which does not proceed to loss of consciousness.

Acute Epilepsy.—A term applied to eclamptic convulsions.

Alcoholic Epilepsy.—Convulsions due to alcohol, directly or indirectly.

Auditory Epilepsy.—A form of epilepsy due to disease of the middle ear.

Cortical Epilepsy.—A synonym of Jacksonian epilepsy.

Diurnal Epilepsy.—In which the fits occur in the daytime.

Gastric Epilepsy.—Arising from some irritations in the alimentary canal.

Hemiplegic Epilepsy.—Generally of syphilitic origin, in which half of the body or one limb only is convulsed.

Hysterical Epilepsy.—The same as hystero-epilepsy.

Idiopathic Epilepsy.—The ordinary form of epilepsy in which no organic cerebral lesion is found after death.

Acute Infantile Epilepsy.—A synonym of infantile convulsions.

Intestinal Epilepsy.—An old term for infantile convulsions, depending on irritating matters in the intestines.

Jacksonian Epilepsy.—Attacks of an epileptic character, confined to one leg, one arm, or a group of muscles.

* Vol. I, p. 449, 1892.

Larvated Epilepsy.—Same as masked epilepsy.

Maniacal Epilepsy.—Acute insanity due to epilepsy.

Masked Epilepsy.—An English term, corrupted from the French (*épilepsie larvée*) and applied in cases in which there is only partial loss of consciousness.

Matutinal Epilepsy.—Epilepsy in which the seizures take place only in the early morning.

Mental Epilepsy.—The same as masked or psychic epilepsy.

Nocturnal Epilepsy.—In which the fits occur only during the night.

Partial Epilepsy.—In which the seizures are confined to a part of the body only.

Psychic Epilepsy.—Periodic occurrence of psychic disturbance, or sensory illusions, without muscular involvement.

Reflex Epilepsy.—Epilepsy resulting from reflex irritation of nerves; as, for example, neuromata; cicatrices; tumors that involve peripheral nerves; intestinal worms; ovarian or uterine irritation.

Spinal Epilepsy.—A term proposed by Marshall Hall for the *grand mal* or complete form of epilepsy, in which sphagiasmus, odaxismus, and laryngismus succeed each other in regular order.

Provoked Spinal Epilepsy.—A term for the condition known as ankle reflex.

Symptomatic Epilepsy.—Epilepsy caused by or a symptom of some other disease.

Syphilitic Epilepsy.—Epilepsy due to syphilitic infection, inherited or acquired.

Thalamic Epilepsy.—A form of epilepsy supposed to be due to a lesion in the optic thalamus. Special sense auræ are supposed to oftenest occupy this form.

Traumatic Epilepsy.—Epilepsy due to an injury to the brain, whether the lesion is a coarse one or not.

Vasomotor Epilepsy.—A type of epilepsy in which the course is supposed to be due to some implication of the vasomotor centers, without affecting the brain, muscular spasms being absent.

I am aware that the foregoing was not written as a distinct classification, but rather for the purpose of

defining; but while it defines, it specifies at the same time the cause in numerous instances, such as *alcoholic epilepsy*, *gastric epilepsy*, *auditory epilepsy*, *renal epilepsy*, *intestinal epilepsy*, etc., and being alphabetically arranged, might erroneously be held to include a full list of causes so far as they are known.

I can also see much in it that would greatly puzzle the student of epilepsy, in the use of different terms to express the same set of conditions; as, for instance, when *abortive epilepsy* is stated to mean "an epileptic seizure which does not proceed to a loss of consciousness." This is naturally enough confounded with *Jacksonian epilepsy*, for here the complete loss of consciousness is not the rule. Or, again, it would be easy to confuse the *masked epilepsy*, *mental epilepsy*, and *psychic epilepsy* this distinguished author gives; the one characteristic running through them all being a sudden, pronounced change in mentality, or temporary loss of mind, without any other symptoms whatever.

Gowers * presents no classification based on etiology. One is chiefly impressed with the positiveness of his views on similar heredity as being the chief cause of the disease, for he goes so far as to pointedly assert: "Epilepsy is an inherited disease."

At the same time, he also refers to other causes, including cortical injury, reflex irritations that cause symptomatic fits only in the beginning, toxic agencies, and emotional shock, studying each under the proportionate influence they exert both as proximate and remote factors in etiology.

In 1870 Echeverria † wrote as follows:

"Epilepsy, like paralysis, is not a morbid entity existing by itself, but a manifestation of manifold derangements disturbing the nervous system and

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* "Epilepsy and Other Chronic Convulsive Diseases," 1901.
† Echeverria on "Epilepsy," 1870, p. 10.

giving rise to definite, inseparate conditions—imme-
diate cause of the convulsive paroxysm—that remain
the same whatever be the occasional origin of epilepsy.''

"No other malady exhibits a wider range in its
etiology.   There is scarcely a disease deranging the
human frame in which epileptiform convulsions might
not happen as an accident or essential phenomenon,
and it may be safely set down as a truth of great
importance that the numerous conditions capable of
inducing epilepsy give to each of its species a charac-
teristic impression that will ever prevent conforming
their individual symptons to any typical case, or find-
ing any specific cure for every instance of the disease.
To establish the peculiar morbid conditions influencing
its development, to discriminate the general from the
local circumstances, in order to arrive at a rational
and successful treatment, is the fundamental question
in the study of epilepsy.''

It is of interest to bring forward the views of this
writer to show that while the widespread and protean
causes of epilepsy were so fully appreciated a third
of a century ago, nothing has yet been done in the
way of classifying clinical types on an etiologic basis.
Echeverria himself made no effort in this direction.
He appears to have made use chiefly of the terms
grand mal and *petit mal* in speaking of seizure types,
giving accounts of these in connection with Marshall
Hall's so-called *spinal system*.

Nothnagel makes use of the following simple classi-
fication:

1. "Epilepsies in which the classical paroxysms
make their appearance with coma and general con-
vulsions.   (*E. Gravior, haut mal.*)
2. "Epilepsies in which paroxysmal loss of con-
sciousness alone occurs, the spastic element for the
voluntary muscles at all events being absent.   (*E.
Mitior, petit mal.*)
3. "Epilepsy in which, with inconsiderable loss of
consciousness, partial twitchings occur in regions of
certain muscles, whereby in the most various ways

a transition is effected between the cases mentioned under 1 and 2.

4. "To these we add the irregular forms of seizures and the epileptoid states."

Turning to the French writers, in the very excellent works of Féré and Voisin but little is to be found in the way of classification founded on etiology. Indeed, the *Definitions* offered by Féré, while failing to convey to the student much information as to what epilepsy is, show very comprehensively how wide a range of pathologic conditions must underlie its many forms.

Féré says: "As a matter of fact, we must look upon epilepsy as a group of symptoms which may appear in greater or lesser number in the course of very different pathologic conditions, sometimes under one form, sometimes under another. To say that there is only one true, essential epilepsy which occurs without appreciable cause, seems to me to be no more admissible than to claim that there is only one true angina pectoris, which has for its cause the contraction of the coronary arteries and false anginas of toxic, hysterical, and other origin. Epilepsy applies solely as a symptom-complex and may be general or partial."

In the above quotations there is a truth so sound, so full of scientific accuracy, and so worthy of universal recognition that I venture to repeat them in substance that they may make a more lasting impression.

"Epilepsy is not a morbid entity," says Echeverria.

"Epilepsy is a group of symptoms, due to different pathological conditions," says Féré.

These, it is true, do not help *per se* in our search for a classification founded on etiology, but they do show that the causes are numerous and that any classification on this basis must recognize part, if not all, of them.

The designations of the different forms of epilepsy offered by Voisin are no more systematically arranged in their causal relationship than are those of other distinguished writers.

It is scarcely worth while to pursue this phase of the subject further. Enough has been said to show that the time is not yet ripe for a satisfactory etiologic classification, as the causes of epilepsy are so numerous and so varied. For the present, the best etiologic classification must be based on causes in the order of their importance, and which are now generally accepted as underlying the different forms of the disease. In doing this, mention must be made of two facts which stand out more prominently than all the rest, namely:

1. That epilepsy is a condition or disease dependent on nervous or degenerative disease in the parent more frequently than on any other single cause.

2. That fully 80 per cent. or more of all cases of epilepsy begin before the twentieth year.

These two facts, which bear the prestige of well-nigh universal recognition, form the basis of a practical classification for present use.

GROUP I.

*Embracing the Epilepsies most Common to the Infantile Period, beginning at Birth and Ending with the Third Year.**

INFANTILE INHERITED EPILEPSY.

Similar Heredity. — Under this head are included cases in which the fundamental elements of epilepsy itself were handed down from parent to child—direct inheritance—as distinguished from indirect inheritance, in which epilepsy in the child is due to some other disease than epilepsy in the parent, such as alcoholism or insanity.

Dissimilar Heredity. — Under this are included all cases in which epilepsy is due in the main to such diseases as alcoholism and insanity in the parent; and in which there is transmitted to the child a general instead of a specific tendency to a particular disease.

* The fixing of this and other age periods is more or less arbitrary, as we shall see later on when we come to study the ages at which definite epilepsies are apt to develop under the stimulus of certain causes. The *practical* importance of the age-grouping scheme is its chief recommendation.

The origin of epilepsy under the causes above are due in the majority of cases to some exciting cause which close study may reveal, while in other cases the epilepsy comes on in strongly predisposed children without any exciting cause that we can discover.

INFANTILE ACCIDENTAL EPILEPSY. { Including pre-natal causes, such as inherited syphilis, birth accidents, asphyxiation, and injuries from forceps; the specific fevers, such as scarlet fever and whooping-cough; the convulsions of dentition in suitable cases; emotional shock due to fright, and cerebral palsy.

In these cases hereditary influences may or may not assist in producing the convulsions, the immediate or exciting causes being in themselves qualified to bring on the attacks.

INFANTILE TRAUMATIC EPILEPSY. { Including cases due to mechanical injuries inflicted on the surface of the body in a manner to affect the integrity of vital internal functions, mostly in the form of injuries to the brain, and possibly to injuries of peripheral nerves causing reflex convulsions in the beginning.

INFANTILE IDIOPATHIC EPILEPSY. { Including all cases in which, after careful and repeated examination, we fail to find any of the causes named in the three forms above.

We may ascribe the origin of some cases under this head to heredity, if such influences can be found; while others must stand as idiopathic epilepsy alone.

GROUP II.

Embracing the Causes of Epilepsies Common to Childhood and the Early Life Period, beginning with the Fourth Year and Ending at about the Twentieth Year.

ACCIDENTAL EPILEPSY.... { Embracing all cases in which the disease is due to the infectious fevers, such as scarlet fever; to peripheral irritations of various kinds that excite and eventually produce habit epilepsy; to emotional shock and to cerebral palsy.

TRAUMATIC EPILEPSY .... { Embracing all cases in which epilepsy is the direct result of a mechanical injury to the brain.

DEVELOPMENTAL EPILEPSY { Embracing cases in which the stress of puberty, acting on a weakened organism, brings to light the nervous disorder to which the individual is predisposed. Cases of delayed heredity fall under this head in a good many instances, the stress of the period serving as the exciting cause.

IDIOPATHIC EPILEPSY .... { Embracing all cases in which, after careful, repeated examinations, we fail to find any cause, proximate or remote, inherited or acquired, sufficient in kind or degree to bring on the attack.*

GROUP III.

Embraces all Epilepsies in which there were no Convulsions Prior to the Twentieth Year, and which Occur from any Cause after that Period.

ACCIDENTAL EPILEPSY.... { Embraces all cases of syphilitic infection; all cases of peripheral irritation; cases due to the infectious fevers; and those due to ovarian or uterine irritations of all kinds and degrees, the latter at first producing reflex convulsions only, but which, if unchecked, may degenerate into a severer form of the disease.

TOXIC EPILEPSIES .... { The toxic causes embrace alcohol, which is more prominent than all the rest combined in this group; the intestinal toxemias; poisoning from lead (a form of exceeding rarity), and all others due to chemical poisoning of any kind.

It may appear inconsistent to place syphilitic infection among the accidental causes and alcoholic poisoning among those due to toxic causes, but this is done because the latter is more a disease due to design than to accident, drinking itself being the essential act that creates the epilepsy.

TRAUMATIC EPILEPSY .... { Embracing cases in which epilepsy is the direct result of any mechanical injury to the brain. This form of cause is the same through all the age periods.

IDIOPATHIC EPILEPSY .... { Embracing all cases in which, after careful, repeated examinations, we fail to find any cause, proximate or remote, inherited or acquired, sufficient in degree or kind to bring on the disease.

SENILE EPILEPSY .... { Embraces all epilepsies due to senile changes, mostly to those in the vascular system, which become prominent any time after the fortieth year, though not usually until the sixtieth or after. This class might properly be included with those due to accidental causes, but is sufficiently distinctive to class it alone.

* The question might properly be raised as to why many of the convulsions of early life should not be classed as eclamptic. Undoubtedly they should be at first. We follow Gowers ("The Infantile Causes of Epilepsy," "Clinical Lectures," p. 205) in here including cases in which eclamptic convulsions later passed into true epilepsy.

Plate I.

SENILE EPILEPSY.

Epilepsy due to senile changes, particularly arteriosclerosis, is not infrequently met with after the forty-fifth to fiftieth years and more frequently after the sixtieth year. The first typical grand mal convulsion occurred in Z. R. at eighty-nine years. Similar attacks continued at intervals of a few weeks until his death when he was nearly ninety-three. He had a marked general atheromatous condition, noted on autopsy, and was subject to infrequent periods of mild psychic disturbance that lasted from two to three days. He suffered no permanent mental impairment, had a remarkable memory, and exhibited none of the stigmata so often noted in subjects of essential epilepsy.

We might now take up each of the foregoing heads, divide and subdivide the causes that make up each; but that would only tend to greater confusion, in addition to creating a drift toward a special name for the epilepsy that follows each specific cause. As it is now, it includes the principle of the cause only, not all its manifestations.

METHOD OF EXAMINING FOR EPILEPSY.

It is comparatively rare for the average student or physician to witness any considerable number of epileptic convulsions in their entirety; that is, to see the prodromal, the active convulsive stage, the end of the fit, and to study its effects on the patient immediately after, and during the inter-paroxysmal period, which may be of a few minutes' or of several months' duration.

It is *the diagnosis of the type of the convulsion itself*, however, that is of the most importance. The outlined examination scheme that follows is based on this fact. Other aids to diagnosis are fully considered in the chapter under that heading.

EXAMINATION SCHEME OUTLINED.

The examination of every case may be divided into four parts as follows:

1. The family history.
2. The personal history.
3. The history of the epilepsy.
4. The personal examination of the patient.

The fact that epilepsy is so often an inherited disease makes systematic inquiry into the family history important, and this inquiry to be of the greatest value should be conducted under an orderly arrangement of questions similar to the following, which is transcribed in part from the official records in use at the Craig

3

Colony, and modified to better meet the requirements of the general practitioner.

The Patient's Family History.

1. Father: Living or dead? Age? If dead, ascertain the age at death, and the cause of death.
2. Mother: Living or dead? Age? If dead, ascertain the age at death, and the cause of death.
3. Father's father: Age? If dead, state age and cause.
   Father's mother: Age? If dead, state age and cause.
4. Mother's father: Age? If dead, state age and cause.
   Mother's mother: Age? If dead, state age and cause.
5. Number of children in the family: Boys; girls; the patient's number in line of birth.
   Number of children: Living? Dead?
6. Did the patient's brother, sisters, father, or mother ever have epilepsy? "Spasms"? "Fainting spells"? Nervous prostration? Hysteria? Insanity? If so, at what age, in what way, and how long did it last?
7. Have any uncles or aunts or other near relatives ever had any of the above diseases or any other form of nervous disease? If so, state which relative and describe the condition.
8. Was the patient's father or mother ever given to intemperance, and to what extent?
9. Have either ever suffered from severe headaches at fixed times?
10. Did the father or mother ever have rheumatism, tuberculosis, scrofula, syphilis, or blood-poisoning of any kind?
11. Was there epilepsy, or insanity, or any other nervous diseases in the grandparents?

The Patient's Personal History.

1. Date of birth: Year. Month. Present age.
2. Was the patient born at full term? If premature delivery, state at what month and all facts regarding the same?

3. Was labor normal in duration?
4. Was delivery natural or instrumental?
5. Was the patient injured in any manner during delivery?
6. Was the mother injured or frightened previous to the birth of the child?
7. Weight of the patient at birth? Was the baby strong or "puny"?
8. Did the patient have "spasms" or convulsions immediately after birth?
9. Was the patient *paralyzed* in any way at or immediately after birth?
10. Was the patient nursed by the mother or fed artificially?
11. Was the patient, as a baby, subject to "indigestion," "pain in the stomach," or prolonged "fits" of crying?
12. At what age did teething commence? Was it difficult, and how?
13. Did the patient have any "spasms" or "convulsions" during teething?
14. Did the patient have fits of crying during the night, or "start in its sleep," or have "night terrors" during infancy or early childhood?
15. At what age did the patient commence to walk?
16. Did the patient have any difficulty in learning to walk, or show any evidence of being weak in one leg, or of being paralyzed?
17. Did the patient show any evidence of "rickets" or of "scrofula"?
18. Was the patient given cod-liver oil or tonics as a child?
19. Did the patient suffer from swollen glands about the neck?
20. Did the patient ever suffer from frequent attacks of "nose-bleed" coming on without any apparent cause?
21. Did the patient have an accident or injury of any kind, especially about the head, in infancy or early life? Was it ever necessary for an operation of any kind to be performed?
22. Ascertain if the patient had any of these diseases and whether they left any "mark": Scarlet

Fever? At age of ——. Measles? At age of ——. Diphtheria? At age of ——. Whooping-cough? At age of ——. Meningitis? At age of ——.

HISTORY OF THE PATIENT'S EPILEPSY.

1. At what age did the patient have first convulsions or epileptic seizures?
2. Have them described as fully as possible by the person who witnessed them, if that can be done.
3. Ascertain the supposed cause of the first attack.
4. How soon after the first attack did the second occur?
5. What was supposed to be the cause of the second attack?
6. Find out how often the attacks have occurred during the past two or three years, and whether they have changed any since the first attack, and if so, in what way.
7. Get a description of the last attack, as full as possible.
8. Does the patient have a "warning" of any kind that an attack is coming? If so, ascertain its nature and constancy.
9. Does the patient have more than one kind of attack (that is, severe and mild attacks)? If so, get a description of each.
10. Find out which arm or leg, or side of the face, or other part of the body, is *first and most often affected*, and whether consciousness is apparently lost at each attack.
11. Ascertain if the patient remembers what took place during the attack, and how long after it is over before he is able to resume work engaged in before the attack came on.
12. Has the patient ever received any serious injury, burn, fall, fracture, or dislocation during an attack?
13. Is there "false sight" or "false hearing" at any time, especially just before or after a convulsion?
14. How has the patient's memory been affected by the disease, and has there been a change in disposition. Is there much irritability?

15. Has there ever been any paralysis of any kind following seizures? If so, was the patient seriously ill at the time the paralysis appeared or was first noticed, and what was the nature of the paralysis? Has it become better or worse since first noticed?

16. Has there ever been loss of speech following seizures?

17. What is the greatest number of seizures or "spells" the patient has had in twenty-four hours?

18. Were these seizures or "spells" all alike? If not, how did they differ?

19. Does the patient ever have a large number of attacks in one or two days accompanied by high fever and marked prostration?

20. How often does the patient have a "series of attacks"?

21. When did such a series occur last?

22. Is the patient getting better or worse, and why?

23. What form of treatment has the patient received?

24. Is there any defect of sight, or hearing, or speech?

25. Is there any tendency toward gluttony, or unnatural appetite for any particular article of food or drink?

26. Does the patient sleep well?

27. Does the patient show any tendency to destroy things, or to injure himself or others?

28. Is the patient obedient and does he respect the authority of those who look after him?

29. Can the patient read and write, and does he possess special aptitude for any particular line of occupation, such as music, drawing, arithmetic, etc.?

30. Is the patient especially uncontrollable before or during seizures?

31. Is the patient cleanly in habits and tidy in dress?

32. Has the patient any vicious habits?

If we can get satisfactory answers to the foregoing questions, we will be able to form a very fair estimate of the case, though to base a diagnosis and outline a plan of treatment on this alone, would be unjust to the patient and an unscientific procedure on the part

of the physician, for nothing can give so complete information as a thorough personal examination, repeated as often as necessity demands.

Confirmed epileptics often have a nomenclature of their own. Some who would not know the meaning of a seizure, know of "spells"; others have only "fits," others "convulsions," "weaknesses," "dizzy spells," "turns," etc. This should be borne in mind in history-taking.

THE PERSONAL EXAMINATION.

The Physiognomy of the Patient.—We can sometimes make a tolerably certain diagnosis of epilepsy from inspection of the patient alone, two pronounced facial conditions making this possible: one, which is the far more common, the unsightly eruption due to excessive bromid intoxication; the other, the existence on some prominent part of the face or head, such as the point of the chin, the supraorbital ridge, or the occiput, of a segregation of scars caused by repeated falls by which the same place is injured each time.

The bromic acne is a less sure guide than the scars, for very similar eruptions may be due to other causes. These are especially apt to occur in favorable subjects about the age of puberty.

The *facies epileptica* of which we are accustomed to hear, is of exceeding rarity if it is held to include facial expressions or conditions to be regarded as pathognomonic of epilepsy.

The only class of cases to which it should be unfailingly applied would be those of the *grand mal* type, in which the convulsions throw the patient or cause him to fall in such a manner as to strike the same part of the body each time, eventually producing a mass of scar tissue in which it is possible to distinguish repeated injuries. This, combined with a large degree of mental impairment, and a profuse bromic acne,

make up the only true *facies epileptica*.   These stig-
mata of the disease are, in part at least, being rapidly
destroyed by modern scientific treatment.

The illustrations presented (Plates 13, 14, 15, 16, 17,
19, and 20) show these conditions very clearly.

The mental condition should be carefully studied in
every case, for much may be learned from this alone
as to the type and frequency of the disease.

In comparatively recent cases in which the epilepsy
is due to organic disease, the effects on the mind may
be very slight, and any pronounced impairment may
be postponed for years, and in rare instances never
appear in a way to attract the especial attention of
the patient or of his friends.

In other cases, in which the cause is largely hereditary,
or the disease is idiopathic, and in which the convul-
sions began very early in life, rapidly and progressively
growing worse, the mind may be entirely destroyed in
two or three years, or even in less time.

The effects of epilepsy on the mind may be classified
under five heads:

In the first the effect is substantially *nil*, the patient's
mental condition being *good*, save momentarily during
a mild seizure.

In the second a slight impairment, usually a loss of
memory, is observed; the mental condition in these
cases being *fair*.

In the third the degree of mental impairment has
progressed a step further, affecting the patient's judg-
ment, personal ambition, and volition, the patient here
being *feeble-minded*.

In the fourth there is a still more pronounced con-
dition of infirmity, the patient being robbed of nearly
all brain power.   He is still able to look after his
bodily wants, as a rule; although in some cases this
power, too, is impaired or destroyed.   These are cases
of *imbecility*.

In the fifth there is total lack of brain and body power. The loss of mental integrity is complete. This form, *epileptic dementia*, is the lowest condition of all, save the lowest types of *epileptic idiocy*.

Memory, without exception, is the first faculty of the mind to suffer. Fully 90 per cent. of all the patients that have ever come under the writer's observation evidenced some loss of memory. Many cannot remember recent events, although they can distinctly recall things that happened before they had the disease, even as far back as thirty or forty years. Recent attacks seem to disorganize and destroy the impression of recent events without affecting the memory of things learned long ago.

Unless we are careful we may sometimes be led into the error of mistaking the effects of long-continued use of the bromids for essential mental impairment due to the disease. The point of differentiation is the restoration of the mental faculties on the withdrawal of the bromids.

A number of instances of temporary mental aberration, closely resembling primary dementia, and due solely to the excessive and unguarded use of the bromid salts, have come under my notice during the past fifteen years. In two cases death was the result.

THE PHYSICAL EXAMINATION.

No lesion in an epileptic, or in a person suffering from epileptiform convulsions, is too slight to be looked for, and it is of the utmost importance that a thorough physical examination should be made in every case. It is unnecessary to describe here the various instruments of precision required for the purpose, since they are the same as those used ordinarily in neurologic diagnosis. But mention will be made of what may be found as possible causes of the disease.

Forms of paralysis are quite common in epileptic

subjects, but they are not always conspicuous. The apparent absence of lameness or other disorders of motility, in any part of the body should not warrant the neglect of stripping the patient and carefully examining the whole body, measuring the muscular force, noting the circumference of the legs and arms, testing all the reflexes, superficial and deep, in addition to carefully examining the sight, hearing, and the general sensory conditions all over the body, in the search for heightened or lessened sensorial perceptions and anesthetic or hyperesthetic areas.

Care should be exercised not to confound forms of temporary or exhaustion paralysis due to recent convulsions with paralysis due to more fixed organic causes, such as cerebral palsy.

Nutritional disturbances are sometimes met with in epilepsy. In view of the part the toxemias of gastric and intestinal origin seem destined to play in future as contributory or specific causes in the production of certain forms of epilepsy, they demand our careful attention.

Gluttony among certain epileptics is proverbial, and frequently the regulation of dietetic habits is one of the first indications of treatment.

The heart and lungs during epileptic convulsions suffer in greater proportion than any of the other organs, with the single exception of the brain. Consequently it is important to carefully examine the heart and lungs in every case, special inquiry being made for tuberculous tendencies or diseases, for asthmatic affections, for angina pectoris and other diseases of the heart, especially those of atheromatous type, which in old persons sometimes cause symptomatic epilepsy of a very distressing type.

Other irritative lesions that may produce *habit epilepsy* are found among the following: Tumors, central and peripheral, the latter occurring as neuromata,

the former occurring in any form that affects the brain; cicatrices; foreign bodies; reflex irritation of carious teeth; vesical and renal calculi; nasal and nasopharyngeal growths; ocular defects, chiefly of astigmatic type; helminthes of various kinds; and otitis.

The reproductive organs call for more than a superficial examination. First of all, the urine, especially collected just before and after the seizure periods, should be thoroughly examined, chemically and microscopically, in the search for renal and bladder disease, and for an increase in urinary deposits due to excessive changes in metabolism.

Urethral strictures, adherent prepuce, ovarian and uterine irritation, and the various forms of displacement should be carefully searched for, effort being made to show if there is an excess of convulsions about the menstrual epoch.

The thermometer is of value in establishing epilepsy, provided it can be used just before and, especially, immediately after the attacks, when some elevation of temperature may be noted in more than half of all cases. In the chapter on Diagnosis the use of this instrument is referred to in detail.

THE STIGMATA OF DEGENERATION.

The epileptic offers a wide field for the study of the stigmata of degeneration. Under careful scrutiny his characteristics in this respect fully come up to those of other defective classes, particularly the insane.

"These stigmata," says Peterson,* "are vices of functional and organic evolution. The deviation from the normal may be in the way of excesses or arrest of development. They must be distinguished from the deficiencies or deformities produced by accident at birth or by disease."

* "Mental Diseases," Church and Peterson, p. 613, 1899.

Plate 2.

Palate with Gothic arch (Peterson).

The hip-roofed palate (Peterson).

Peterson presents an elaborate classification of the various degenerative indices, to which the reader is

Fig. 1.—Chemocephalus or flat-headedness.
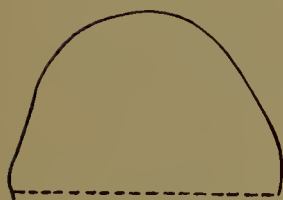
Fig. 2.—Oxycephalus or steeple-shaped skull.
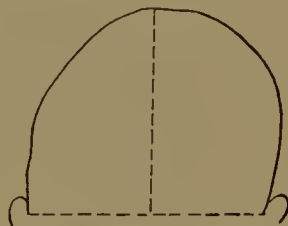
Fig. 3.—Plagioce-phlus or obalique deformity of the head.

Vertex

Postr.—»--»Antr.

Antr.

Postr.

Vertex

Right___Left
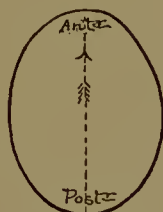
ANTERO-POSTERIOR   CIRCUMFERENCE   TRANSVERSE

Fig. 4.—Microcephalic contours.

Postr.——»»----»--Antr.

Antr.

Postr.

Right___Left.
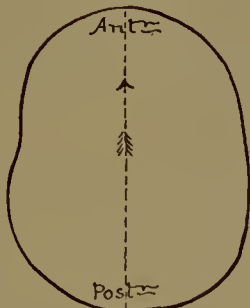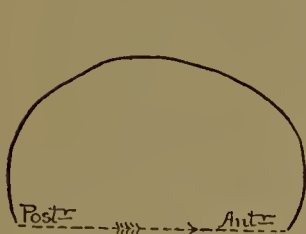
Fig. 5.—Hydrocephalic contours.

referred for more complete information on the subject. This classification is under three heads, and in substance is as follows:

Anatomic Stigmata.
        Cranial anomalies.
        Facial asymmetry.
        Deformities of the palate.
        Anomalies of the teeth.
        Anomalies of the tongue and lips.
        Anomalies of the nose, eyes, ears, and limbs.

Fossa ovalis.
Fossa cymbæ.
Fossa concha.
Incisura intertragica.
Fossa scaphoidea.
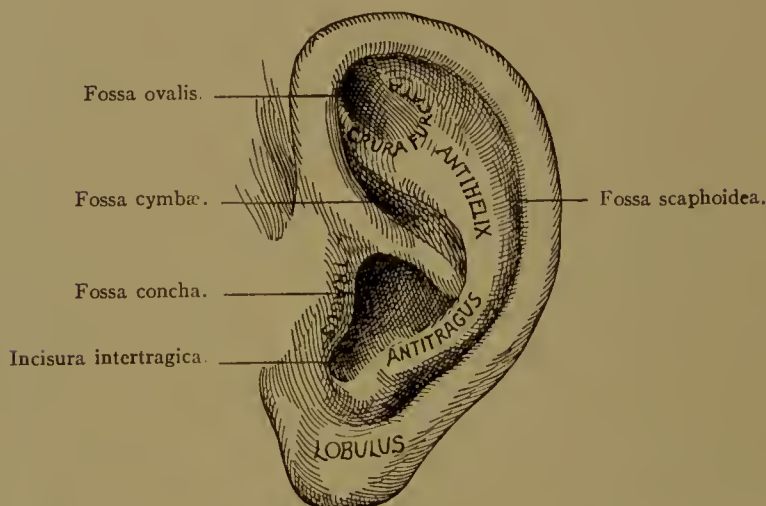
Fig. 6.—Normal ear.

Fig. 7.—Excessive length of ear; fusion and distortion of helix, anthelix, antitragus, and lobule (Peterson).
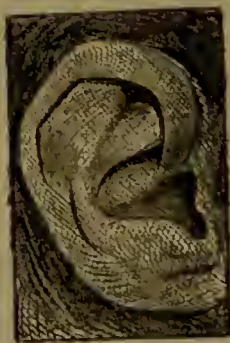
Fig. 8 —Triplication of crura furcata; malformed helix and antitragus; absent lobule (Peterson).

        Various body malformations.
        Anomalies of the skin and hair.
Physiologic Stigmata.
        Anomalies of motion:
                Tics, tremors, etc.

Plate 3.

Low grade imbecile epileptic, showing "Morel" ears marked by abnormal development of the helix, antihelix, fossa scaphoidea, and crura furcata, causing the folds of the ear to seem obliterated. Such ears are smooth, larger than usual, prominent, and have a thin edge.

Plate 3 a.

MARK SEVERE SEIZURES WITH X. PUT D AFTER DAY SEIZURES.

Month					Month					Month					Month				
1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
6	7	8	9	10	6	7	8	9	10	6	7	8	9	10	6	7	8	9	10
11	12	13	14	15	11	12	13	14	15	11	12	13	14	15	11	12	13	14	15
16	17	18	19	20	16	17	18	19	20	16	17	18	19	20	16	17	18	19	20
21	22	23	24	25	21	22	23	24	25	21	22	23	24	25	21	22	23	24	25
26	27	28	29	30	26	27	28	29	30	26	27	28	29	30	26	27	28	29	30
31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___	

MARK MILD SEIZURES WITH O. PUT N AFTER NIGHT SEIZURES.

Month					Month					Month					Month				
1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
6	7	8	9	10	6	7	8	9	10	6	7	8	9	10	6	7	8	9	10
11	12	13	14	15	11	12	13	14	15	11	12	13	14	15	11	12	13	14	15
16	17	18	19	20	16	17	18	19	20	16	17	18	19	20	16	17	18	19	20
21	22	23	24	25	21	22	23	24	25	21	22	23	24	25	21	22	23	24	25
26	27	28	29	30	26	27	28	29	30	26	27	28	29	30	26	27	28	29	30
31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___	

MARK "DIZZY SPELLS" WITH DOT (•)

Month					Month					Month					Month				
1	2	3	4	5	1	2	3	4	5	1	2	3	4	5	1	2	3	4	5
6	7	8	9	10	6	7	8	9	10	6	7	8	9	10	6	7	8	9	10
11	12	13	14	15	11	12	13	14	15	11	12	13	14	15	11	12	13	14	15
16	17	18	19	20	16	17	18	19	20	16	17	18	19	20	16	17	18	19	20
21	22	23	24	25	21	22	23	24	25	21	22	23	24	25	21	22	23	24	25
26	27	28	29	30	26	27	28	29	30	26	27	28	29	30	26	27	28	29	30
31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___		31	Total Mild___		Total Severe___	

Facsimile (reduced) of a folder card designed by the author, to be carried by the patient for keeping a record of epileptic seizures. All attacks for twelve months can be entered on it, the character of each attack being noted. A dietary for epileptics is printed on the back of the original.

Anomalies of sensory functions:
 Deaf-mutism, neuralgia, migraine, and various ocular defects.
Anomalies of speech:
 Mutism and stammering.
Anomalies of the genito-urinary functions:
 Sexual irritability, impotence, and sterility.
Anomalies of restraint or appetite:
 Uncontrollable desire for food, liquor, and drugs; lessened resistance against disease; retardation of puberty.
Psychic Stigmata.
 All degrees of mental impairment:
  Insanity, idiocy, imbecility, feeble-mindedness, precocity, marked eccentricity, moral delinquency, and sexual perversion.

Deformities of the *cranium, palate, face,* and *ears* are among those most commonly observed, the accompanying illustrations (Figs. 1 to 8 inclusive) showing the more common types.

THE KEEPING OF SEIZURE RECORDS.

In addition to the seizure data kept by the physician, it is well to place in the possession of the patient a card so arranged that he or his friends may readily enter on it the date and character of every convulsion. The card should also contain the patient's name and address, for use in case of accident.

The possession of such a card may prevent the patient considerable annoyance should he commit unreasonable acts while in an epileptic or automatic state for which he is in nowise responsible.

CHAPTER III.

FREQUENCY, AGE, SEX, RACE, AND OCCUPATION.

Proportion of Epileptics to the General Population. The Age of Greatest Development. The Influence of Heredity on Age. Comparative Frequency in the Two Sexes. Race. Occupation.

FREQUENCY.

It is difficult to reach an accurate conclusion as to the frequency of a disease so unobtrusive in its manifestations as epilepsy not infrequently is, and so universal in extent, geograpically speaking, as to apparently exempt none of the races of the earth.

It cannot be found that any estimate of the proportion of epileptics to the population at large was made until after the epileptic began to be a public charge in some of the countries of continental Europe, about half a century ago—in France, in 1849; in Germany, in 1867.

Chiefly through reports * made to the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics in this country in 1901 and 1902, a fair estimate may be made of the prevalence of epilepsy in this and foreign countries.

Kollé gives the proportion of epileptics in Switzerland as 1 to 750.

Kovalevsky states that the proportion in Russia is 1 to 2000, as a general rule, there being 80,000 in that country all told, adding that the proportion in the great wine-drinking provinces of Caucasus is much in

* "Transactions of the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics," 1901 and 1902.

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excess of this; while Shoutelwort and others state that one person out of every 1000 in Russia suffers from epilepsy.

Pelmann estimates the number in Scotland at 15 in every 10,000, or 1 to every 750, making 6000 in Scotland alone.

Vernet, basing his figures on those made by Lunier in 1881, places the proportion in France at 9.2 for every 10,000 inhabitants, making the enormous number of 33,225 in the French Republic.

Jules Morel estimates the number of epileptics now under family care in Belgium at from 3500 to 4000, exclusive of the large number in the Colony at Gheel.

Basing his estimate on statistics gathered in many States in this country, including New York, New Jersey, Connecticut, Massachusetts, Virginia, Illinois, Indiana, Texas, and others, Peterson estimates the ratio of epileptics to the population at large in the United States at 1 to 500, making the tremendous aggregate of from 140,000 to 150,000.

I do not feel that this estimate is too high. Indeed, it is scarcely high enough, for there are many cases of the milder forms that go unrecognized, and others still which, through feelings of pride, or other motives, are kept concealed, so that they are never included in any census that is made.

The author's personal knowledge of many cases under both these heads leads him to believe that the estimate of one epileptic to every 500 inhabitants of the country at large is not too high. As compared with insanity, it is safe to say that epilepsy is a little more than half as common as that disease.

AGE.

Epilepsy is a disease of all ages. On this point all observers agree. In latent form it may be in the infant at birth, or it may not appear until senile

changes are well established, at seventy years or beyond. Observers also agree that it is far more common during the first twenty years or so of life than during any period thereafter.

Gowers * sums up his age studies of the disease as follows:

"More than a quarter of all cases begin under the age of ten years; nearly half between ten and twenty; about a seventh between twenty and thirty; a sixth between thirty and forty; about $2\frac{1}{2}$ per cent. only between forty and fifty; 1 per cent. only between fifty and sixty; after which half of 1 per cent. only occur. Seventy-four per cent. of the total number of cases begin under twenty."

A closer analysis is also made by the same author in which he gives the number for each year from 1 to 71, a summary of which is as follows:

A large number of cases, no less than $13\frac{1}{2}$ per cent. of the whole, began during the first three years of life.

In this group no case of simple infantile convulsions is included; only such as began in infancy and continued as chronic epilepsy.

Of about a third all that could be learned was that they began in infancy. In making up the table these were distributed through the first three years of life in the same proportion as presented by the two-thirds in which the exact date of commencement could be ascertained. The number of cases is largest at the first year; falls rapidly to the third year of age, then more slowly until five, when the minimum for the early period of life occurs.

From this there is a considerable rise at seven, the commencement of the second dentition, then a fall at eight, from which the number increases slowly at nine and ten, rapidly at twelve until the maximum is

* "Epilepsy and Other Chronic Convulsive Diseases," 1901.

reached at fifteen or sixteen, at which 166 and 167 cases occurred. From this time there is rapid fall to twenty-one, after which only a few cases began each year.

The table that follows is made up from a series of 1302 cases which came under the writer's observation during a period of eight years, and shows the number of cases that occurred each year from 1 up to 70:

Year.	Cases.	Year.	Cases.	Year.	Cases.	Year.	Cases.
1	163	19	33	37	7	55	1
2	67	20	26	38	5	56	1
3	48	21	27	39	3	57	2
4	38	22	16	40	3	58	0
5	30	23	15	41	2	59	0
6	43	24	6	42	5	60	1
7	48	25	7	43	3	61	0
8	54	26	12	44	3	62	1
9	48	27	10	45	4	63	0
10	56	28	10	46	3	64	0
11	48	29	6	47	3	65	2
12	71	30	5	48	0	66	6
13	56	31	6	49	1	67	1
14	84	32	6	50	1	68	0
15	59	33	4	51	2	69	0
16	43	34	5	52	1	70	1
17	44	35	9	53	0		
18	32	36	4	54	2		

It is observed from this table that the greatest number occurred during the first year, that they then regularly declined until the sixth year, when the number rose again during the second dentition, to drop off very slightly during the ninth year, when 48 only occurred; after which a third increase began, reaching a maximum at fourteen, when 84 cases occurred, followed by the final decline to the seventieth year, only 67 cases out of the 1302 having occurred after the thirty-fifth year.
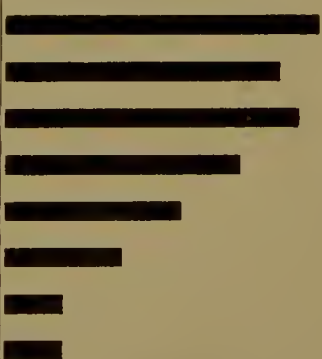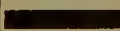
By changing the yearly table into decimal periods we find the number and percentage for each age period to be as follows:

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Under 10 years 499	38.5 per cent. of the total number.	
10 to 20 " 566	43.5 " " " "	
19 to 29 " 125	9.5 " " " "	
29 to 39 " 54	4 " " " "	
39 to 49 " 27	2 " " " "	
49 to 59 " .... 11 ⎫	1.75 " " " "	
59 to 69 " .... 12 ⎭		

It may now be recalled that the classification scheme presented in the previous chapter was largely based on three age periods, the first including the first three years; the second, the fourth to the twentieth, inclusive; the third, the remainder of life.   We can better appreciate the practical importance of this after observing that 21 per cent. of all cases began during the first period, and 62½ per cent. during the second, leaving an average of but a fraction over 1 per cent. for each year thereafter.

Our studies furthermore show that more than 83 per cent. of all cases begin under the twentieth year as compared with 7⅘ per cent. given by Gowers.

The table below shows very comprehensively the tendency of the disease to decline with the increasing age of the patient, the studies not being carried beyond the fortieth year because of the rarity of the development of the disease *de novo* after that period.

Under 5 years.	████████████	346—26½ per cent.
5 to 9 years inclusive.	███████████	249—19⅛ "
10 to 14 " "	████████████	318—24⅖ "
15 to 19 " "	██████████	178—13⅔ "
20 to 24 " "	███████	71— 5½ "
25 to 29 " "	█████	43— 3⅓ "
30 to 34 " "	███	30— 2¼ "
35 to 39 " "	███	22— 1⅔ "

THE INFLUENCE OF HEREDITY ON AGE.

Although the study of hereditary influences properly belongs under etiology, it is of interest to allude to it here, so far as it has bearing on the age periods that aid in bringing its influences to light, and the detailed statement that follows shows the age, up to the forty-fifth year, at which all hereditary influences are most active, also their comparative effect on the two sexes at the same age:

Year.	Heredity or no Heredity.	Males.	Females.	Year.	Heredity or no Heredity.	Males.	Females.
1	Heredity.	49	39	24	Heredity.	2	2
	None.	51	28		None.	0	0
2	Heredity.	28	10	25	Heredity.	3	0
	None.	21	15		None.	3	2
3	Heredity.	17	7	26	Heredity.	2	0
	None.	14	7		None.	5	2
4	Heredity.	19	8	27	Heredity.	2	2
5	Heredity.	6	7		None.	3	2
	None.	12	5	28	Heredity.	2	0
6	Heredity.	5	10		None.	4	4
	None.	21	8	29	Heredity.	4	0
7	Heredity.	14	12		None.	0	0
	None.	15	7	30	Heredity.	3	0
8	Heredity.	22	7		None.	0	0
9	Heredity.	15	10	31	Heredity.	0	0
	None.	19	3		None.	3	0
10	Heredity.	16	13	32	Heredity.	2	0
	None.	22	5		None.	3	0
11	Heredity.	16	5	33	Heredity.	0	0
	None.	20	12		None.	0	0
12	Heredity.	17	19	34	Heredity.	2	0
	None.	15	19		None.	0	0
13	Heredity.	10	15	35	Heredity.	2	0
	None.	10	15		None.	3	4
14	Heredity.	27	18	36	Heredity.	0	0
	None.	21	12		None.	2	0
15	Heredity.	18	3	37	Heredity.	3	0
	None.	14	12		None.	2	2
16	Heredity.	8	11	38	Heredity.	2	0
	None.	17	5		None.	2	0
17	Heredity.	9	8	39	Heredity.	0	0
	None.	12	13		None.	4	0
18	Heredity.	6	7	40	Heredity.	0	0
	None.	8	8		None.	2	0
19	Heredity.	12	2	41	Heredity.	0	0
	None.	5	9		None.	0	0
20	Heredity.	3	4	42	Heredity.	0	0
	None.	12	7		None.	3	0
21	Heredity.	9	5	43	Heredity.	0	0
	None.	6	4		None.	0	0
22	Heredity.	4	3	44	Heredity.	0	0
	None.	5	3		None.	0	0
23	Heredity.	4	3	45	Heredity.	0	0
	None.	2	2		None.	0	0

This table shows conclusively that inherited causes play their most important rôle in early life. Eighty-

eight out of 163 cases which occurred during the first year were ascribed to heredity. During the second year 38 out of 67 were due to the same. During the third year 23 out of 48, the same proportion (about 50 per cent.) keeping up throughout, for as the number of cases increases during the second dentition and puberty periods, the hereditary influence is increased in corresponding ratio.

After the twentieth year the influence of heredity rapidly declines, and disappears almost completely by the fortieth year, there being but a few isolated cases after that in which such a factor could be traced.

In all studies bearing on the age at which epilepsy develops, and the influence of heredity at different ages, the further investigation is carried the more firmly is the student convinced that epilepsy is essentially a disease of early life, with heredity as an influential or contributing cause in a large proportion of all cases.

RATIO OF FREQUENCY IN THE TWO SEXES.

There exists a considerable diversity of opinion among some of the most noted writers on epilepsy as to its comparative frequency in the two sexes.

Gowers is authority for the statement that women suffer from epilepsy rather more than men, the proportion being thirteen of the former to twelve of the latter.

Féré * says: "Nearly all authors admit that epilepsy is more frequent in women." The statistics, however, upon which these writers base their opinions are not available for comparison.

Hare † seems to place little reliance on some of the figures made by well-known authors covering this point, and mentions Esquirol and Moreau as believing

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* "Epilepsy," "Twentieth Century Practice of Medicine," Vol. x, p. 618.
† "Epilepsy; Its Pathology and Treatment," p. 102.

with Gowers that epilepsy is more common among women than among men, adding: "Other figures made by Englishmen collected by the writer combat the views of these three writers by placing the greater number of cases among men."

"Boyd," says the same author, "in studying 145 cases found a third more men than women.'

Althaus * collected in all over 54,000 cases, dividing them into five-year periods, beginning in 1847 and ending in 1871, 28,690 of them being men, 25,482 women. Hare regarded this showing as conclusive.

"No especial influence," says Osler,† "appears to be discoverable in their relation, certainly not in children. Of 433 cases in my tables, 232 were men and 203 were women, showing a slight preponderance of the male sex. After puberty, unquestionably, if a large number of cases are taken, the men are in excess."

Echeverria studied 306 cases of epilepsy, 130 of them being men, 176 women; adding that the disproportion in the two sexes was probably due to some extent to the amount of hospital accommodations available for each.

English writers generally are credited by this author with reporting more male than female epileptics.

The United States Census Bureau reports deaths from epilepsy in the United States for the "census years" (every tenth year) from 1860 to 1900, as follows:

Census Years.	Total Deaths from Epilepsy.	Males.	Females.
1860	501	284	217
1870	1414	778	636
1880	2157	1215	942
1890	2367	1316	1004
1900	3326	1915	1411

* "Diseases of the Nervous System," p. 222.
† "The Principles and Practice of Medicine," 1902, p. 1094.

Acording to these figures there occurred in the five census years only, a total of 9718 deaths from epilepsy in the United States; 5508 of them were men and 4210 were women, an excess of 1298, or 25 per cent. of the former over the latter. In 1870, 2.9 per cent. of the deaths were due to epilepsy; in 1880, 2.9 per cent.; in 1890, 2.8 per eent.; in 1900, 3.2 per eent.

The apparent increase in the number of deaths may be accounted for on the basis of more carefully compiled statistics.

Wharton Sinkler is of the opinion that epilepsy is more common among men than among women. Out of 1204 cases at the Orthopedie Hospital and Infirmary for Nervous Diseases, Philadelphia, 548 were men, 476 women.

Mailhouse (1902) reported 542 in Connecticut; 315 men and 227 women.

In a total of 324 cases in one hospital in Virginia reported by Dr. Drewry, 221 were men, 103 women. This observer adds: "In the statisties I collected a few years ago regarding epilepties in Virginia, I found that men were decidedly in excess of women."

The report of the New York State Lunaey Commission for 1900 shows that between 1888 and 1900, 616 deaths occurred from epilepsy in the State Hospitals for the Insane; 392 men and 224 women.

During a period of eight years, from February 1, 1896, to February 1, 1902, histories of a total of 1582 well-authenticated cases of epilepsy came to the author's notice, 951 of which were men, and 631 women.

All of these were seeking relief in a speeial institution, and the fact that the capacity of the institution was greater for men than for women may have caused a smaller number of women applicants to appear; yet it is not likely that a sufficient number were kept from applying to account for the great diserepaney between

the two sexes in the actual number who did apply. In a total number of 68,040 cases mentioned above, 36,865 were men, 31,175 women.

It is the author's belief that epilepsy is more frequent among men than among women, in the proportion of 20 men to 16 women in every 100 cases. The apparent failure of the English writers to indicate the epilepsies due to alcohol and syphilis which occur most often in men in middle life may partly account for their views on the preponderance of the disease in the female sex.

Through the period during which heredity plays its most active part there is less difference in the number between the two sexes than later on, but after that the fact that men suffer more accidents which cause traumatic epilepsy, more often have syphilitic infection of the brain, and are more commonly given to indulgence in alcoholic drink, are the chief reasons why epilepsy is more frequent in the male sex.

RACE.

In the same way in which epilepsy makes no distinction in the social status or condition of those it affects, it makes equally as little so far as race is concerned.

Naturally the greater number of cases occurring in any one country under usual conditions, would be among the people of that country, and merely to give some idea of the universal character of the disease, I mention the following countries, exclusive of the United States, as contributing to cases I have studied since 1895: Germany, England, Russia, Ireland, Canada, Austria-Hungary, Sweden, Italy, Switzerland, Japan, France, Roumania, Arabia, and Belgium.*

* Among the races of other countries known through reports to the National Association for the Study of Epilepsy and the Care and Treatment of Epileptics, to suffer from Epilepsy, are: Turkey, India, Australia, Chili, and Brazil. "Epilepsy is very common in all parts of Brazil" (Hoaelburg)

Out of a total of 1322 in which the race was definitely ascertained, 57 in all, 41 men and 16 women, were of the Jewish race, while two were American Indians, one of each sex, and both under fifteen years of age.

Searcy, who has had exceptional opportunities for studying epilepsy and insanity in the negro race in the Southern States, says:*

"There are about thirteen times as many insane negroes under state care now in Alabama as there were thirty years ago. In 1870 there were 33 insane negroes in our hospital; in 1880, 71; in 1891, 241; in 1900, 451."

"It is," he adds, "a generally expressed opinion that insanity and epilepsy are rapidly increasing among the negroes. Many lines among them are degenerating and epilepsy and insanity are but indications and results of it. The Africans in America came out of their servitude an inherently improved people, mentally and physically. While they were slaves there was little deterioration among them, consequently, little insanity."

Viewed solely from its scientific and hygienic side, the lessons taught by the history of the negro race in the South under slavery, when their habits, appetites, and tendencies to dissipation were largely regulated and controlled, and under freedom from slavery, when all these hurtful tendencies were allowed to run full riot, are of undoubted value to the student who is looking for the great causes that degrade and destroy the mental and physical life of the people who practise them.

OCCUPATION.

So far as I have been able to ascertain, occupation has no influence in the production of epilepsy.

While it is true that we find the great majority of cases among representatives of certain callings, it is

* "Report of the Alabama Bryce Insane Hospital," 1902.

due to the relatively greater number engaged in such callings and not to any specific influence exerted by the form of occupation itself.

We found 702 epileptics out of 1322 following one or the other of these vocations: Mechanic, farmer, laundryman, seamstress, painter, porter, merchant, musician, messenger, peddler, barber, clerk, printer, nurse, engineer, railway conductor, sailor, bartender, plumber, bookkeeper, tailor, lawyer, teacher, dentist, clergyman, student, photographer, mason, shoemaker, drummer, letter-carrier, cooper, fireman, flagman, draughtsman, upholsterer, newsboy, teamster, coachman, watchmaker, and others.

Six hundred and twenty, nearly 50 per cent., had no occupation of any kind. The occupations of housewife or domestic furnished the greatest number of any single calling; 195, equal to about 15 per cent., coming under these heads.

Laborers stand second in frequency; farmers, third; mechanics, fourth; while clerks and students stand fifth, with the same number, 50, each.

Under this heading, were it within the province of this work, we might study to advantage the ethic and economic side of the effects of epilepsy—a disease which often impairs or completely destroys the usefulness of its victim in the commercial and social world, without any appreciable impairment of mind or body, the knowledge of the presence of epilepsy, no matter how mild or infrequent the attacks may be, sufficing to make the epileptic's presence undesirable.

But such considerations hardly fall within the scope of our present studies.

CHAPTER IV.

ETIOLOGY.—PART I.

Heredity: Similar and Dissimilar. Consanguinity. The Causes Active in Early Life. The Infectious Fevers: Scarlet Fever; Typhoid Fever; Measles; Diphtheria; Malaria. Emotional Shock and Fright.

WE may assemble the causes of all epilepsies primarily under two heads:

Predisposing.

Exciting.

Under the former will be studied the comparatively few factors that prepare the individual to acquire the disease, but which may not actually produce it; under the latter, as many as possible of the numerous factors that actually produce epilepsy with or without the aid of influences that predispose to it.

In the majority of cases both predisposing and exciting causes play a part and are complemental to each other. This well-known fact we may illustrate in this way: Given an individual in whom epilepsy may be induced under the influence of some irritation of which the climax—*i. e.*, the point at which it causes the seizure to appear—is represented by 100. If there are already present 60 of these points from, we will say, *a predisposition due to heredity*, there will remain but 40 points to be supplied by some exciting cause to bring the disease to light; this exciting cause being, perhaps, one of the specific fevers, some kind of toxemia, an emotional shock, an acute attack of indigestion, trauma, some reflex irritation, or numerous other causes that will be fully described later on.

On the other hand, if we say there are but 40 points

of a predisposing cause present, and the "seizure point" is the same as before (100), it will require 60 points of the exciting cause instead of 40 to develop the convulsion.

In other words, epilepsy, etiologically considered, is often a symptom-complex dependent in the great majority of cases upon two variables; either *a maximum of inherited influence combined with a minimum of exciting cause, or a minimum· of inherited influence combined with a maximum of exciting cause.*

The fact that heredity, both similar and dissimilar, plays such an important part in the production of epilepsy is so universally recognized that I will omit all but a few authoritative opinions on this point:

"Epilepsy is an inherited disease." *

"There are few diseases in the production of which inheritance has more manifest influence, and the traceable influence is always far less than that which exists."†

"The significance of this (heredity) at the present day no longer needs to be proved by statistics and special instances; it is a fact noticed and conceded by all observers."‡

"Hereditary transmission of epilepsy may be direct or indirect; that is to say, it may be affected through the antecedents or through the collaterals. . . . . Dissimilar heredity has long been recognized in epileptics." §

"Heredity is the great factor in mental and nervous diseases. In epilepsy it also plays the chief rôle." ‖

"Heredity is the most potent of any single influence."**

* Gowers, Allbutt's "System of Medicine," p. 763.
† Gowers, "Epilepsy and Other Convulsive Diseases," p. 3.
‡ Nothnagel, "Cyclopedia of the Practice of Medicine," Ziemssen, p. 201.
§ Féré, "Twentieth Century Practice," Vol. x, p. 616.
‖ Voisin, "L'épilepsie," 1897, p. 6.
** Dana, "Nervous Diseases," ed. 1897, p. 464.

"The children of epileptics are frequently insane, idiotic, or hysterical, and the descendants of an insane person are often epileptic, idiotic, or insane, or their epilepsy passes into insanity, or epilepsy supervenes upon idiocy."*

Whatever skepticism one might feel about the in-influence of heredity when entering upon the systematic, intimate study of hundreds of cases, much, if not all, of it will be dispelled before the end of many years.

All close students of nervous diseases come sooner or later to feel the force of Maudsley's statement concerning insanity and crime when he says:

"They are as much manufactured articles as are steam engines and calico printing machines. They are neither accidents nor anomalies in the universe, but come by law and testify to causality, and it is the business of science to find out what the causes are, and by what law they work."†

As potent as are the influences of heredity in insanity and crime, they are none the less so in epilepsy.

But heredity is not the only cause; indeed, as we shall presently see, it is far from it. My chief purpose in bringing it so clearly into the foreground is because it is the factor we should command ourselves to search for first in each case that comes under our care. If it is not present, all the better, for the prospects of amelioration or cure will be proportionately brighter; if it is present, we should know it, for the knowledge will materially help to guide us in treatment, especially if the treatment is surgical and designed to remove bodily the proximate cause of the disease from the brain.

Some of the most notable failures in the surgical treatment of epilepsy that I have known were due to failure to consider heredity when it constituted the

* Ireland, "Mental Affections of Children," 1900, p. 14.
† Gorton, "Ethics of Pauperism and Crime," 1902, p. 204.

sole ascertainable cause. At the same time, we should not feel that hereditary influences are fatal to amelioration or cure. This would be a pessimistic view altogether unwarranted by the plainest teachings of experience.

It has come to be regarded that no matter what the cause, if the mental condition of the patient remains inherently unimpaired, improvement is always possible, and even cures can sometimes be wrought when they are least expected.

It will be profitable now to go back and take up the study of causes somewhat in the order named in the classification on pages 30 to 32.

HEREDITY.

PREDISPOSING CAUSES ACTIVE IN EARLY LIFE.

Similar.	*Dissimilar.*
In which the disease appears in the child because a predisposition to it was acquired from the parent who had the same disease.	In which the disease appears in the child because a predisposition to it was acquired from the parent who had some disease of the nervous system other than epilepsy, such as alcoholism or insanity.

Both similar and dissimilar heredity from any point of view constitute predisposing causes only; neither, in all probability, can actually create the disease, although the immediate exciting agent, in some instances, may be exceedingly difficult or impossible to determine, at least in the present state of our knowledge.

And why? The answer to this at once carries us over into a consideration of the probable pathologic changes involved in epilepsy, and since we shall have frequent occasion for referring to this portion of the subject before we come to the chapter on Pathology, where it is exhaustively dealt with, we will briefly

discuss the changes now thought to underlie the epi-
leptic state.

It is generally agreed that it is the function of the
nerve cells in the gray matter, the *cortex* of the brain,
to store up nervous energy, and it is the function of
this energy to be ready for release the moment any
call is made upon it. Irrespective of the source from
which this energy is derived or the manner in which
it is acquired, it must be taken up and stored by the
nerve cells, which then stand charged with it, so to
speak, like a bucket full of water, in a state of the
most delicate equilibrium.

The process of securing, storing, and releasing
nervous energy in a normal way may seem simple
enough at first glance, if viewed merely as an abstract
physical problem; but such is not the case, for it
constitutes the sum total of life itself, and we have
absolutely no knowledge of the intricate principles
of the vital processes involved; the only two things
that seem reasonably clear being:

First, that nervous energy is secured from nutritional
processes, is appropriated, and in some way stored up
by the nerve cells.

Second, the gradual discharge or giving off of this
energy to meet the immediate and varying demands
of the entire organism for all the purposes of life.

The nerve cells' income of force or energy is thus
ordinarily kept equal to the outgo of the same force.
In normal nervous action any discrepancy in these
two quantities is not sudden, sharp, and destructive,
at least not to the extent it is in epilepsy, but is
gradually discharged through legitimate physiologic
channels under inhibitory processes designed to regu-
late the supply in a natural way.

If the power of the nerve cell to *inhibit* itself or be
inhibited is impaired in any way, either through a
deficiency or weakness that existed at birth, or through

some disease that appeared after birth, it will release its energy in an irregular, spasmodic way, which neurologists agree as constituting the basic cytologic changes that give origin to the epileptic convulsion.

Now comes the question, Why do nerve cells do this? In what way is their natural ability to store up and retain nervous power impaired?

On this deeply interesting point science cannot yet speak with any degree of conviction. It assumes that the fault lies either in the structural malformation of the cell, it being too small to do the work required of it, in its imperfect insulation, or in the over-production of nerve force. All, or any, of these conditions are assumed to be present.

It seems difficult to understand how a nervous system, frail, unstable, degenerate, and always weak in its structural composition and force, can rapidly store up, discharge, and re-accumulate nervous force of the degree and kind required to create epileptic manifestations unless its power of inhibition is impaired or destroyed.

Some neurologists appear to think that the cause of the essential epileptic expression—the convulsion itself—is to be explained wholly on the ground of ineffective inhibition.

Nevertheless, it seems reasonably fair to assume that the *delicacy of adjustment* between the chemical relations—the tendency of the atoms to unite in fresh and closer combinations and release their latent energy—is very great, so that the slightest deviation from the normal relation between the molecules overturns equilibrium, when a quick combustion and sudden release of energy occur.

The causes of epilepsy in early life may then be said, because of all this, to be more largely predisposing than exciting, the predisposition lying in an inherently faulty central nervous system that was as much a part

and parcel of the child at the moment of its birth as was the shape of its ear or the color of its eyes.

Now, add to this inherited fault some exciting cause, some form of "stress," and the cause is complete—the individual is ready for epileptic manifestations.

PROPORTION OF CASES DUE TO SIMILAR HEREDITY.

In one series of 1070 cases that came under the writer's observation—660 men and 410 women—105 of the men and 73 of the women had the disease because of the same disease in the parent.

The number in the men constituted 15 per cent. of the total, while the number in the women amounted to 17 per cent. of the total. Combining both men and women, the percentage of the total number in all cases was 16. It appears from this that more women than men have epilepsy as a direct inheritance from the parent.

It would be interesting to compare these figures with the results of other observers, but it cannot be found that any one else has tabulated results which single out *similar heredity* from *dissimilar heredity*, such as alcoholism and insanity tend to confer.

If Gowers included in his studies on heredity only those cases in which "like comes from like" his results are numerically greater than those obtained by the writer, for he says:

"Particulars as to heredity and age at commencement were noted in 2222 cases, in 888 of which heredity existed, or about 40 per cent."

In the absence of any designation to the contrary, I incline to think that all hereditary factors, similar and dissimilar, were included by Gowers; but if this is true, our results compared with his will again differ, for, combining all inherited influences, including epilepsy, insanity, alcoholism, tuberculosis, and other diseases, I found a total of 56 per cent., or 16 per cent. more than that stated by Gowers.

It has been claimed that inherited epilepsy always developed before the age of twenty years, but by referring to the table on page 51, in which is shown the number of cases that developed in the two sexes, from the first year up to the forty-fifth, it will be seen that this is a mistake. It is true that the decline in heredity at about the twentieth year is very marked, yet we note that 28 cases among men and 12 among women, 40 in all, began between the twentieth and thirtieth year; while 9 occurred among men between the thirtieth and fortieth year; cases occurring in either sex after that age were not ascribed to hereditary influences.

It is worthy of note that no women, after the thirtieth year, had the disease as a result of heredity of any kind, while in nine men it was the ascribed cause. This seems perfectly reasonable when we recall that the exciting causes in men about this age, such as alcohol, syphilitic infection, and various forms of trauma, are vastly more common than among women.

AN ILLUSTRATION OF SIMILAR HEREDITY.

The following is as clear a case of similar inherited epilepsy as I have ever known, and is reported as a typical illustration of the cause under discussion:

C. E. M. came under my observation in 1898, being then nineteen years of age. She had been an epileptic six years, the cause being ascribed to "menstrual disturbance."

Her family history was not good. Her father had rheumatism; her mother and half-sister died of cancer. The patient "cried almost constantly," and "had had night terrors as a child." She had a convulsion during the first dentition and a second one at the age of two years, having been free from them after that up to the age of thirteen, when they began to appear at each menstrual epoch. At eighteen years she had status epilepticus, which came very near

5

destroying her life. The individual attacks were of the severest kind.

Eight months after she came under care, she gave up treatment and returned to her home, having improved greatly in that time.

In the fall of 1902 she re-appeared under another name, having been married about two years, and I learned that a year after her marriage she had a baby—a boy—who seemed well and strong up to the third month, when, without any appreciable cause, he had a severe convulsion, followed two months later by a second, and a month later by a third. The physician now having the baby under his care reports it as suffering from established epilepsy.*

The mother of this child unquestionably inherited a depraved constitution, and had she not been obliged to pass through the disturbing physiologic epochs that bring to light the weak points in such constitutions, she might have gone through life free from epilepsy.

But this was not to be, for the first dentition, acting as an exciting cause on a constitution made ready for disease by a faulty heritage, provoked the first convulsion, which was followed by others made easier of development through the beginning of the "habituation of the epileptic habit," established by the first fit.

The mother is now twenty-three years old and has been an epileptic ten years; yet, because the brain has a predisposition to epilepsy only—does not possess the genuine epileptic dyscrasia, the convulsions appearing as the result of an irritation outside the brain—she still has a good mind, and should she follow the usual course of such cases she may continue to have the full use of all her mental faculties, even though she should live to reach the age of sixty or seventy years.

Now, contrast this very probable picture with the

* Shortly after this was written, the baby died of cerebrospinal meningitis.

prospective future of the child if it had lived. Born of a mother burdened with a strong predisposition to epilepsy, it did not wait for the active crisis of the first dentition epoch to excite the convulsive habit, but began to have distinct epileptic seizures at three months; and had death not occurred, it would undoubtedly have become an epileptic idiot at an early age.

The cause of epilepsy in the mother was dissimilar heredity, plus the stress of the first dentition; while the cause in the child was similar heredity plus, may be, some slight stress, in all probability nutritional in kind, but the exact character of which we are unable to identify.

PROPORTION OF CASES DUE TO DISSIMILAR HEREDITY.

Alcohol.—The same group of 1070 cases that yielded 16 per cent. in which the cause was similar heredity, contained 111 men and 51 women, 16 per cent. of the former and 12 per cent. of the latter, in which the dissimilar hereditary factor of alcoholism in the parent led to epilepsy in the child.

The explanation of the greater preponderance of epilepsy due to drink in men is undoubtedly due to the fact that the drink habit is more marked in this sex, a fact our experience tends to confirm through having seen so many cases among middle-aged men due to this cause, while we recall but a single case in which it was clear that drink directly brought on the disease, or laid the foundation for it, in a woman.

In estimating the number of epileptics in different portions of Russia, Kovalevsky writes as follows:*

"Caucasus is a country of grape and wine-making. The drinking-water from the mountain rivers is bad, but the wine is good. The natives of Caucasus quench

* "Transactions of the First Annual Meeting of the National Association for the Study of Epilepsy," 1901, p 168.

their thirst, not with water but with wine, and the
wine is no light one.   It contains from 5 to 15 per cent.
of alcohol.   Wine-drinking is so common in Caucasus
that no one considers it inebriety.   Everybody knows
what a high percentage of epilepsy is caused by the
abuse of alcoholic beverages.   I have spent the sum-
mers during the last fifteen years in Caucasus, where
I have a medical practice drawn from a large district,
and in no other place have I had so large a proportion
of epileptics among my patients.''

Of such vital importance and so full of interest is
the whole question of the effects, both immediate and
remote, of alcohol on the human race, that I quote
literally from a recent Scottish authority, whose valu-
able work deals with pathologic factors active in
uterine life.   Under a study of fetal alcoholism,
Ballantyne has this to say:

"Another question concerned with the effects of
alcohol upon antenatal life remains to be considered,
namely, the *dystrophic* and *teratological* results.   With
regard to epilepsy developing after birth, there is a
great deal of evidence that parental alcoholism is an
immediate and powerful etiologic factor, Féré, Combe-
male, Leter, Lancereaux, Montpellier, and others
having written on this subject; while Bourneville,
who studied 2554 children admitted to the Bicêtre
and Fondation Vallée—2072 boys and 482 girls—all
of them suffering from idiocy, epilepsy, imbecility, or
hysteria, found that 1053 of them were the offspring
of drunken parents, 933 having drunken fathers, and
80, drunken mothers.

" Sullivan found that out of 219 children of alcoholic
mothers who lived beyond infancy, 4 per cent. of them
became epileptics; a very high proportion as com-
pared to the frequency of the disease in the general
population.''

Insanity.—It appears singular that insanity should

constitute a less predisposing cause to epilepsy than alcoholism, but such is the case.

In 660 men and 410 women, insanity in the parents was found in 49 men and 42 women, or 7 per cent. of the former and 10 per cent. of the latter. In this case the conditions, so far as proportions go when compared with alcoholism, are reversed; there the greater number occurred among men, while here it is the women that suffer most.

Combining both sexes, we have a total of 91 cases, or about 7 per cent. of the entire 1070, due to dissimilar heredity induced by insanity.

Tuberculosis.—In taking up tuberculosis as a possible predisposing factor in epilepsy, we are treading on delicate ground. So far as I have been able to ascertain, there has never been any attempt to specifically connect the tuberculous diathesis in the parent with epilepsy in the child.

We have simply assumed that its presence in the parent gives the child a grant, a predisposition, a something that seems to invite epilepsy in preference to any other nervous disorder. Apparently we have only to study the tables of heredity to find this true.

In the same 1070 cases just considered under alcohol and insanity, a distinct tuberculous history in the parents was found in 101 men and 50 women, making 15 per cent. in the former and 12 per cent. in the latter, altogether 151, equal to 14 per cent. of the entire number.

Account was taken of the pulmonary form of tuberculosis only, there being no case known in which the disease affected the central nervous system, or its meninges.

This percentage seems extraordinarily high, standing second to epilepsy itself, in which the percentage was 16, and next to alcohol, in which it was 15, and

it can only be accounted for in this way: Tuberculosis in the parents either predisposes to epilepsy in the offspring, or it is so common as to make it appear influential when it may be only an incidental affection. Let us note the opinion of others in this respect:

"It has been claimed," says Hare, "by those who ought to know, that a family history of phthisis is a cause of epilepsy." He adds that such a belief exists among both English and American authors, and quotes Hamilton as having 230 cases in a series of 980, "phthisically inclined."

Doubt, however, is cast over the entire statement because he says that Hamilton's practice was among "a depraved, half-starved class in a great city in which consumption and epilepsy might well walk hand in hand."

Nothnagel and Gowers assert that phthisis does not influence the production of the disease, but that they are simply associated in the same individual.

"Phthisis," says Gowers, "was once thought to be connected with the disease by some hereditary mechanism, and the cases of phthisis to be heard of in the families of epileptics are many. But the ratio is not found to be larger than in the families of those who are not epileptics, and an investigation from the side of phthisis clearly showed that the apparent connection was due to the frequency of phthisis."

My own opinion is that phthisis may grant a predisposition to some forms of epilepsy. If there is well-marked phthisis in the parent at the time of conception, there seems to be no reason why a general tendency to disease may not be given the child.

From studies made by Ballantyne in his most admirable work on "Antenatal Pathology and Hygiene," 1902, pages 206 to 216, the author's views would seem not only to be entirely within reason, but to embrace a scientific fact, for Ballantyne collects much evidence

to show the existence of fetal tuberculosis, and we have only to add to the inherited fatality which undermines the entire stamina a sufficiently powerful exciting cause in order to develop definite diseases of the brain, foremost among them being epilepsy.

THE INFLUENCE OF DIFFERENT HEREDITARY FACTORS ON THE AGE AT ONSET OF EPILEPSY.

It is difficult to obtain data completely reliable on this point, but we are able to present the results of careful study along this line in 1217 out of 1300 cases.

The average age for the beginning of the epilepsy in this number was 12.36 years. Taking 574 cases with neuropathic heredity in which the age was stated, the average age at onset was 11.22 years. Leaving these last-named cases out of consideration and taking the cases without such heredity, the average age at onset was 13.26. Taking the cases with alcoholic heredity, the age at onset was 10.8 years. The same average occurred in cases where insanity was noted in the history. The average age at onset in the cases with epileptic family histories was 10.17 years.

The combination of alcoholism and epilepsy in the history brings the average age at the beginning of the disease down to 9.9 years.

The lowest average age at onset was found in cases which had a combination of alcoholism, epilepsy, and insanity in the history. There were six cases of the kind—too few on which to base any conclusion—but the average for these was 9.5 years. These figures prove that heredity influences the age at onset, and also tend to show that, as a rule, the more unfavorable the heredity, the lower the age at onset.

As a further illustration of the influences of heredity on the age at the beginning of the disease, it may be stated that, while 20 per cent. of the total number of

cases and 17 per cent. of the cases with a neuropathic heredity history began during the first three years of life, over 25 per cent. of the cases showing neuropathic heredity began during the same period. During the first ten years of life 46 per cent. of all the cases began, 43 per cent. of those without neuropathic history and 50 per cent. of those with such history having begun during this period.

Between the ages of ten and twenty years there was no difference. Thirty-eight per cent. of the total number of cases, and 39 per cent. of all cases with or without neuropathic history, began at the same period. Eighty-four per cent. of the total number began during the first twenty years; 82 per cent. of cases in which neuropathic history was denied or lacking, and 89 per cent. in which such history was found, began during this period.

Consanguinity.—I do not recall ever having seen a case in which epilepsy could fairly be ascribed to the intermarriage of blood relations alone.

Gowers states that although consanguinity of parents intensifies the family habit, there is no evidence that it has any influence in producing the disease.

I am aware that statistics collected by some authors in Continental Europe, particularly by Bemis as mentioned by Hare, appear to show that such marriages are more common in that country than in this, and that the results are most unsatisfactory from a health point of view.

Syphilis.—There is no more deplorable fact in medicine than the transmission of syphilis from parent to child. It is most often handed down by the father (Holt, Jacobi, Rotch, Koplik). A physician consulted me about his epileptic child, a boy of eight years, whose disease the father was convinced was due to constitutional syphilis transmitted from himself.

Ballantyne feels that Fournier does not state the

case too strongly when he says: "Syphilis is the essential murderer of those young in years; it is the veritable tomb of infants; it is the cause of death before birth, at the moment of birth, after birth, within the first week, or it may await the first year. Syphilis, alcoholism, and tuberculosis constitute the triad of the contemporaries."

I am unable to present figures that indicate the frequency with which inherited syphilis predisposes to epilepsy, or causes it in any form, essential or unessential, and must be content to state that its power in either of these directions must occasionally be reckoned with.

The writer has under his care at this time a boy of seven years whose epilepsy developed under the stress of the second dentition, who was delicate and sickly from his birth, and whose father—a man of intelligence and position—advanced the idea of the cause of the boy's malady by confessing to syphilis in himself when the boy was born. The cause was further verified by the improvement the patient made under anti-syphilitic treatment.

A colleague of the writer's reported to him a case almost identical with the above.

Other predisposing causes include rheumatism, scrofula, rickets, constitutional anemia, gout, cancer, parental intoxication at the time of conception by such drugs as morphin and lead, diabetes, possibly chorea, and organic degeneration isolated in the ovaries and testes.

THE IMMEDIATE CAUSES MOST ACTIVE DURING THE EARLIER YEARS.

Adhering to the group division under Causes, given on page 31, we come now to study the more active as distinguished from the predisposing causes of epilepsy in infantile life, a period we may for practical

purposes arbitrarily assume to terminate at the end of the third year.

These causes are numerous and include, in addition to others, to be specified in greater detail later on, those of a pre-natal or congenital order, birth accidents, the specific fevers and their sequelæ, the stress of the first dentition, and possibly in rare instances, shock from fright, though the latter is less a factor here than later on in the childhood period.

And just here let me state the distinction held to exist between congenital or pre-natal causes, and those assigned to heredity.   By the former is meant a cause that existed in the child at birth, that originated during intra-uterine life or was acquired at birth, no part of which was given the child as the result of some constitutional imperfection in the parents.

By the latter is meant a cause born with the child and acquired as the result of some constitutional imperfection in the parents.

Cerebral Palsies.—Unquestionably one of the greatest single causes at this early age are the brain palsies which show themselves in the form of hemiplegia, diplegia, or paraplegia, the former being far more common than the two latter combined.

In 1070 patients of all ages at the Craig Colony, many of them so far advanced in age as to have lost all but the faintest trace of the results of a cerebral hemorrhage that occurred in infancy, 116 or 11 per cent. had epilepsy as the result of this cause, 67 of the 116 being men and 49 women; as there was no discrepancy in sex, the seeming inequality is due to the fact that more men than women were studied.

The seat of the lesion in these cases is in the hemisphere of the brain, involving the central motor neurons, and that part of the motor tract which extends from the brain cortex as far as the anterior horn.   This makes such palsies cortico-spinal, in con-

Plate 4.

Showing the attitude and deformity in left hemiplegia associated with epilepsy. The perpetuation of the convulsions in these cases is often due to the cerebral hemorrhage that establishes the paralysis.

tradistinction to those due to disorders of the neuro-spinal neurons, which give rise to the forms of infantile spinal paralysis without involvement of the brain.

Cerebral palsies occur after the third year, but the vast majority of them occur before that time, while a third of them are congenital. When the latter is the case, it may be due to an injury received by the mother during pregnancy, or to an injury inflicted on the child through forceps, or to some other accident attendant upon the parturition period.

It is a well-known fact that during the first two months of life reflex action is insufficiently developed and convulsions which appear in that time are almost always primarily cerebral and due to hemorrhage.

It is difficult to over-estimate the damage that may follow the minutest hemorrhage, especially when it occurs at a period marked by such active growth and development on the part of the brain, as in childhood.*

It is timely at this point to impress the importance of searching examinations for evidences of cerebral hemorrhages in every case that comes before us.

The writer has found more than once distinct evidences of a former hemorrhage in cases in which the true cause of the disease had been entirely overlooked.

In such cases hearsay evidence should never be accepted. It is always best to strip the patient; examine all reflexes, superficial and deep; test the muscular power and cutaneous sensation; observe the manner in which the patient holds or carries himself,

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* The writer recalls the impression this first made on him when listening to a lecture on the results of hemorrhage by Virchow some years ago.

Eight or ten specimens were exhibited to illustrate the damage done to brain tissue through minute extravasations almost imperceptible to any but a well-trained eye.

A week later I asked Hughlings-Jackson what he thought might eventually be found to be the cause of most cases of "idiopathic epilepsy," and he promptly replied, "Small hemorrhages in the brain."

and note his gait; in short, leave no stone unturned that may reveal the presence of the results of a former hemorrhage in the brain.

If the patient can be seen immediately after an attack, or a series of attacks, local muscular weaknesses will declare themselves then better than during the inter-paroxysmal period, for the attack serves to further deplete, and even at times temporarily destroy, the foci of muscular innervation in the brain.

For some time I had under my care a young girl whose epilepsy followed a grave colitis at the age of six years; the severe convulsions occurring at that time produced a cerebral hemorrhage which furnished a permanent basis for epilepsy. Ordinarily she had some drooping of the left shoulder only, but after a series of attacks the left leg and arm suffered an exhaustion paralysis that lasted several days.

Such conditions constitute true exhaustion paralysis, so well studied and described by Clark,* and which will be referred to separately when we come to study more at length the physical effects of seizures of different types.

The following cases clearly illustrate the action of the cause under discussion.

W. B. (1157) first came under observation when he was ten years old. He had been epileptic since the age of nine months. His mother was healthy, his father "intemperate." Birth was normal and the child apparently was strong and robust. He had "prolonged fits of crying when a baby." The first convulsion occurred at nine months and "lasted ten hours, and was due to eating green berries." "The first fit," the parents stated (which was really a serial attack), "settled in his right side." The convulsions that followed appeared about once a month at first. He began to walk at fourteen months, at which time "right hemiplegia was still marked." There was total

* "Archives of Neurology and Psycho-pathology," Vol. II, 1899.

Plate 5.

Right hemiplegia, especially involving the foot, occurring at the age of one year. Epileptic convulsions commenced soon after. Note flat plantar arch left foot. Right hand also paralyzed.

lack of mental development, and his present condition, at eleven years of age, is that of an epileptic idiot, with paralysis of one side.

H. S. (994) was born of middle-aged parents. Forceps delivery. Father healthy; mother, maternal aunt, and sister are epileptic. He had an attack of meningitis at eight months, during which "convulsions lasted three days, followed by left hemiplegia." The patient was fourth in line of birth in a family of seven, all of whom, save himself, are dead, four having been still-born. The patient is now ten years old. His attacks occur about once a week, and he has the mind of an imbecile.

J. R. (980) was born of parents past middle age, both of whom were "markedly tuberculous" at the time. Dentition began at fourteen months and the process was difficult. He had "diphtheria at thirteen months, meningitis at sixteen months, scarlet fever at four years, and whooping-cough at five years." First, convulsions came "just after the meningitis and lasted forty-eight hours." After that they came rapidly, two or three a day. The functions of the right eye and the right ear were defective, and the entire right side was paralyzed. This condition followed the first convulsions.

These three cases are typical of a distinct variety that is unfortunately too common.

While it is the convulsion that brings on the hemorrhage, which in time causes the epilepsy, there must be other causes back of it for the convulsion, and these are numerous.

In the case of W. B. the cause was undoubtedly indigestion, due to things wholly improper for food at the age of nine months, though the convulsion might never have occurred had there not been a predisposition to nervous diseases acquired by the child as the result of alcoholism in the father.

In the case of H. S. the convulsion was again only an exciting cause acting on a strongly marked inherited inclination to some nervous disease, while it

was excited to action by an attack of meningitis—the primary cause being the inherited predisposition; the secondary, the meningitis; the third, the convulsion that laid a substantial organic foundation for a continuance of the fits.

The third case, that of J. R., is almost similar to the one just given; the remote cause here being the tuberculosis in the parents; the secondary cause, the meningitis that occurred at sixteen months; the final and lasting cause, the convulsions that followed this.

The prevention of such cases as these and their treatment at a time when the convulsive habit—when the "recurring tendency constitutes the disease," as Gowers says—may possibly be checked before epilepsy is fully established, are matters of vital importance, fitted to engage the attention of the general practitioner who comes into contact with them first.

Dentition.—While it is true that the stress due to the irritation of difficult dentition is capable of exciting convulsions which may sooner or later pass into true epilepsy, it is equally true that it is impossible to reach a conclusion as to even the approximate number of cases so caused, for the reason that the period of dentition is replete with other causes actively productive of the same results.

The period of dentition begins about the middle of intra-uterine life and ends, visibly, first at about the thirtieth month, and secondly, about the twelfth year.

It has long been the writer's belief that *dentition when severe, and acting on an organism that bears the impress of transmitted weaknesses of a necessary kind,* plays an important rôle in the production of epilepsy in early life.

Let us understand, however, that a single convulsion caused by the stress of abnormal dentition is not epilepsy, though it may be perilously near it in patients possessing a depraved constitution, and that genuine

Plate 6.

Left hemiplegia in a boy of nineteen years. The paralysis commenced in the left thumb at two months, extending slowly until the tenth month, when the entire left side was affected. Epileptic convulsions developed two months after onset of paralysis.

epilepsy should be said to exist only when the convulsive habit has become firmly established.   But even so, the dentition, the active factor which causes the first convulsion, is after all the real cause of the disease.

It is interesting to note the views of other writers on this point.

Gowers says: "A considerable number of cases of epilepsy date from infantile convulsions.   In many of them the first fits occur during retarded dentition, commonly with other indications of the state called rickets, as definite convulsions, or as minor attacks, these persisting to puberty, after which they become definitely epileptiform.

"This suggests that the early convulsions produce a lasting modification of the brain, one facilitating the tendency to discharge at the developmental epoch.

"In about a tenth of the total number of cases of epilepsy some such influence must be ascribed to infantile convulsions.   The popular connection of these fits with the process of teething has probably very slight foundation in fact; a slight local irritation may have an exciting influence, but the chief factor is the general retardation of development."

"Epilepsy," says Féré, "may appear in connection with dental disturbance.   The frequency of convulsions in young infants during dentition is well known.   The opinions of physicians who confine their attention entirely to diseases of children, and who have had a large experience along this line, are perhaps as valuable on this point as are the opinions of neurologists."

L. Emmet Holt * deals in part with the subject as follows:

"Although I strongly believe that the importance of dentition as an etiologic factor in disease has been in the past greatly exaggerated, and although I have formerly held the opinion that simple dentition did

* "Diseases of Infancy and Childhood," 1900, p. 243.

not and could not produce symptoms, within the past four years I have been compelled to change my opinion upon the subject, and I am now willing to admit that dentition may produce reflex symptoms, some even of quite an alarming character. ·

"Speaking from impressions and not from statistics, I should say that in my own experience about half of the healthy children cut their teeth without any visible symptoms, local or general."

Holt then enumerates some of the symptoms produced by difficult dentition, including wakefulness at night; fretfulness during the day; loss of appetite; catarrhal stomatitis; constipation or diarrhea; fever, in ordinary cases running up to 101° F., in extreme cases to 104° F., the latter occurring in delicate or rachitic children; and severe attacks of acute indigestion, finally adding : "Convulsions due to dentition I have never seen, yet I do not doubt that they may occur in rachitic children."

J. Lewis Smith in his classical work * says:

"Among the more common pathologic results of difficult dentition are certain affections referable to the cerebrospinal system. Eclampsia is one of the admitted results. Barrier attributes convulsions in the teething infant to excitement of the nervous system arising from pain which is felt in the gums and to a determination of blood in the dental apparatus, in which reflex the vascular system of the head participates. Difficult dentition must be considered not so frequently a direct as perhaps a predisposing cause, producing a sensitive state of the nervous system or possibly an afflux of blood to the head, and which by an additional trivial stimulus ends in convulsions."

This author also mentions a case in which the appearance of "five incisor teeth in two weeks caused

* "Diseases of Children," 1896, p. 692.

two attacks of eclampsia, all other causes being strictly omitted.''

"Epilepsy," says Voisin,* "is above all the malady of infancy. It makes its first appearance at birth, but generally it is at the moment of dentition that the first manifestations present themselves. In these cases dentition is the 'touchstone' of the hereditary predisposition.''

In a study of the causes of insanity in children by the writer, which appeared in the "Medical News" in 1894, may be found a number of opinions on the mental effects of dentition, a few of which are reproduced in substance here.

Dr. Graeme M. Hammond had "records of several cases in which convulsions due to dentition were followed by true epileptic convulsions.''

Dr. F. Peterson could "recall a number of cases in which epilepsy was due to the convulsions of dentition.''

Dr. T. S. Clouston—the well-known alienist, superintendent of the Royal Morningside Asylum at Edinburgh—said, "I have seen the convulsions of dentition followed by prolonged delirium ending in idiocy, or in true epilepsy, or insanity of adolescence.''

Dr. G. Alder Blumer thought there was "no such thing as a convulsion due to dentition pure and simple.''

Dr. Jacobi wrote: "Every convulsion, ever so slight or short, may produce cerebral hemorrhage, with all its possible results—epilepsy, idiocy, paralysis, and insanity.'' Apparently he did not mean that the convulsions referred to were due to dentition,† but did admit, as stated by the writer, "that the spasms and convulsions of infancy are *serious* manifestations, and if allowed to go unchecked may lead to explosions of genuine epilepsy and later on to insanity.''

* "L'épilepsie," 1897, p. 23.
† "Causes of Epilepsy in the Young," "Medical News," Dec. 13, 1902.

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In eighteen cases of epilepsy that have come under my observation in which the disease began between the sixth month and the end of the second year, the cause was ascribed to "teething," or to "dentition and indigestion"; and it was particularly noted that the family history in the majority of these cases was bad; in some of them extremely so.

Summing up the facts in the case, it seems reasonable to hold that difficult dentition, when acting on an organism suffering from inherited constitutional diseases, like rickets, tuberculosis, alcoholism, epilepsy, insanity, and the like, excites reflex convulsions that in time degenerate into true epilepsy.

The ratio of cases in which simple convulsions may pass into epilepsy is governed entirely by the original stamina of the patient and the vigor with which he is treated immediately after convulsions appear.

Points worth our serious attention are the disturbances of digestion; in fact, the disorders of the entire alimentary canal, so prone to occur in young children at this age, and which may greatly aggravate the effects of difficult dentition, or themselves cause convulsions, through forms of auto-intoxication, erroneously ascribed to carious irritation.

THE INFECTIOUS FEVERS.

We note occasional references in medical literature to the infectious fevers as causes of epilepsy, although we do not find any mention made as to the probable frequency of cases due to such causes.

Scarlet Fever.—This type of fever seems to lead to epileptiform convulsions at first and later on to true epilepsy more often than any other. In a series of 1323 cases, 814 men and 509 women, it was found to be the cause in 25 cases, 18 of which were men and 7 women, the proportion between the two sexes being approximately the same.

The epilepsies due to this cause invariably develop during the essential epileptic age, *i. e.*, before the twentieth year; while the greater number of them appear between the sixth and fifteenth year, a period that embraces the second dentition and the epoch of puberty, both of which strongly aid in the establishment of any nervous disorder in which heredity plays an important rôle.

It also further appears that in the majority of cases it is not the fever itself that excites the convulsions, since they do not generally come on until this has subsided, but the after-effects of the disease, chiefly in the way of *scarlatinous nephritis*.

When the convulsions due to this condition change from general to unilateral, when they have been violent, or appear in series, it is safe to assume that the change in type is due either to a cerebral hemorrhage, to a thrombosis, or to a marked exhaustion paralysis, the results of the former being ineradicable, the latter in some cases susceptible of removal entirely, though not always with the result of checking the attacks.

Another peculiarity of such attacks is this: When a child has scarlet fever, at seven or eight years of age, followed by nephritis and convulsions, the convulsions may disappear entirely in a short while and remain absent for years, to recur under some provocation that seems to excite the original cause into action.

An illustration of this peculiarity is shown in the following case:

E. B. G., whose father had rheumatism and died of "congestion of the brain" at fifty-seven, had a severe attack of scarlet fever when he was eight years old, followed by convulsions which occurred at first daily, then about once a month. On reaching early manhood, the attacks left him for seven years, and he

made a trip to Europe alone during that time. Then they returned, and now, at the age of thirty-nine, they occur from two to three weeks apart. His memory for recent events is poor, though for things that happened earlier in his life it is most excellent.

Besides illustrating a type of the disease in which long remissions occur, it also illustrates one in which the convulsions at first were primarily reflex—due to some disease outside the brain and affecting that organ only in a reflex manner. Such cases are numerous.

Prototypes of the convulsions that appear after an attack of nephritis due to scarlet fever in childhood are found in cases of uremic convulsions which occur later in life, particularly in Bright's disease, when the age of the patient, his lessened powers of resistance, associated with other more serious things, more often combine to destroy life, although there may be cases in the very young in which urinalysis may fail to show uremia as a cause, in which event we must look for something besides the nephritis.

Jacobi * reports: "An example of this kind is now in my hospital ward, a child with nephritis after scarlatina which ran its course four months ago. While practically in convalescence he was taken with *eclamptic* attacks a few days ago. As there was a daily renal secretion of from 500 c.c. to 600 c.c., a percentage of more than 2 of urea, and no indiscretion in his diet, the diagnosis of uremic intoxication was out of the question. This obliged us to inquire into his past, when a history of several unprovoked convulsions of an epileptic character during the past eighteen months was obtained."

In such a case there must have been a strong predisposition to convulsions present, the irritation of the

* "Medical News," Dec. 31, 1902.

nephritis, acting simply as the "touchstone" that brought them to light.

Gowers speaks of the remarkable influence of scarlet fever in causing epilepsy, but doubts that it does it through uremic poisoning. In fact, he goes so far as to say that in most instances any evidence of such disease is lacking, and assumes that the fits are due to some peculiar effects of the scarlet fever poison upon the nervous system.

In the opinion of the writer, fully 2 per cent. of all cases of epilepsy are due to scarlet fever, and we should not dispute creditable statements that seek to place the percentage even higher.

Whooping-cough.—Altogether five cases of epilepsy due to whooping-cough have come under my observation, making about a third of 1 per cent. of the total number.

According to Szabo, of Budapest, in a series of 4591 cases of pertussis, 2695 of them occurred before the end of the third year.

In view of the great frequency and severity of the malady, it seems remarkable that it is not more often the cause of epilepsy, as indeed it may be. It is not uncommon for a child to die during a paroxysm of coughing. The brains of such children are found engorged with blood. Any agency that so greatly and violently increases intracranial pressure in persons of feeble stamina, especially children, may cause cerebral hemorrhage with its consequent results of paralysis and epilepsy.

The convulsions that occur during the paroxysms of whooping-cough may probably be ascribed to the poisonous effects of the excess of carbonic acid in the blood, due to a partial asphyxia.

In the American edition of Nothnagel's "Encyclopedia of Medicine," 1902, under the complications of whooping-cough we find this statement:

"Eclamptic attacks (*convulsions externes*) are not uncommon in children with an inclination to convulsions, or in those who manifest symptoms of hydrocephalus. They occur in mild forms of whooping-cough frequently during teething, yet also as late as the fifth year, and appear usually between the eighteenth and thirty-fifth day of the disease.

"According to some authorities they take place more frequently the more violent the coughing attacks. They are ushered in by excitement or drowsiness, and often again by extreme dyspnea. They occur subsequently to a paroxysm of coughing, or they happen during a free interval, or interrupt the seizure, thereby changing its character, or causing the paroxysms to cease entirely."

Sticker describes the clinical manifestations of the convulsions; how they grow in violence with each repetition, how they decrease in mild cases in frequency and severity, until, finally, all alarming symptoms cease and the child gets well.

In other cases the convulsions that are local in the beginning become general; the child sinks under prostration and dies within the first three days. "Among ten children," he says, "nine are certainly doomed."

In those cases in which the convulsions come on during or just after the coughing paroxysm, and when it is at first local in nature, it is undoubtedly due to some focal irritation in the brain, like a slight hemorrhage, a thrombus, or an embolism.

In those in which the convulsions appear during the remissions that separate the coughing paroxysm, it may be due, as before indicated, to chemical changes in the blood, or to the action of the specific poison of this disease.

All of the cases noted by me occurred in children ranging in age from six months to six years.

A little girl of three years had whooping-cough which was immediately followed by spinal meningitis, attended with convulsions and right hemiplegia; the convulsive attacks became fixed, and five years later they occurred at the rate of from 8 to 10 a month. Such a result as this may follow in any case.

Insolation.—Thermic fever caused the same number of cases that were due to whooping-cough, namely, 4; and we are unable to offer a suggestion as to the manner in which the cause acted, unless we assume that the convulsions were induced by the effects of high temperature, or came from chemical changes in the blood.

All four of the cases studied were males; two, fourteen, one, ten, and the other twenty-five years of age.

A predisposing heredity was present in three, and was extremely marked in one of the younger, there being epilepsy, insanity, and alcoholism in his family. The "father was an alcoholic and a suicide, one paternal uncle an idiot, another an epileptic; the mother had chorea in childhood, and hysteria later on. Another had convulsions, and a sister was an epileptic."

In this case it is not difficult to see that there was ample preparation made in the patient by depraved ancestors to fit him for almost any disease, especially one that came at the epoch of puberty, the period when the sunstroke was received. Alcoholism alone tends to invite insolation, but this boy did not drink, he only inherited an instability from his father, whose stamina was always of a depraved order.

One of the cases was that of a carpenter, who enlisted as a soldier at the age of thirty-two and was sent to Cuba during the Spanish-American war in 1898, where he had a sunstroke followed by convulsions that later developed into epilepsy.

The youngest patient, a boy of ten years, also had a bad heredity; his father was an alcoholic and suffered

from rheumatism; two brothers were habitual drunk-ards and a sister was an idiot.

Typhoid Fever.—In three cases the cause was ascribed to typhoid fever. Two of the patients were women thirty-two and thirty-three years of age; the third was a boy of four years, whose family history was good, while that of both the women was unsatis-factory. In the younger woman it was said there had never been any convulsions prior to the time she had typhoid fever at thirty years of age, which had been followed by "weaknesses," and later by true epilepsy, but this may be doubted. She had a child a year after her marriage at eighteen, who is now an epilep-tic, so this case may not properly be ascribed to the effects of typhoid fever. About the other there seems to be no doubt.

A. C. Brown * reports a case of Jacksonian epilepsy following an abscess of the brain that involved the Rolandic area on the right side. The author states that the abscess was due to an attack of typhoid fever, the same pyogenic organisms which caused the fever having produced the abscess.

Didé † reports cases of epilepsy caused by the in-fection of typhoid fever; in some the convulsions appeared during the fever, and were due to the direct action of the typhoid toxin; while in others they developed after the fever was over, constituting in these cases paratyphoid manifestations, and resulting remotely from cellular changes induced in the nervous system by the action of the poisons generated in the course of the disease.

Cases have also been recorded in which epilepsy existed in conjunction with boils and abscesses about the scalp, the assumption being that they were caused

* "Edinburgh Medical Journal," Sept., 1900.
† "Revue de médecine," Feb. 10, 1899, p. 151.

by a pyemic state, the relief of which cured the convulsions.

Typhus and Yellow Fever.—One case was ascribed to typhus, though the details are not clear enough to give substantiation, and one to yellow fever.   In the latter case, that of a sailor who contracted the disease in a tropical country, the family history could not be obtained.   The patient had no convulsions prior to the age of nineteen, when they followed the attack of yellow fever.

Pneumonia was given as the cause in a boy of seven, whose family history was not good and whose mother had "fainting spells."

Grippe, which so often produces marked morbid changes in the entire nervous system, often leading to insanity, was credited with being the cause in one case, that of a man of fifty, who had long been intemperate and in whom senile changes had set in, which were indicated chiefly by the atheromatous condition of his arteries.

Malarial fever was the cause in two cases; one in a girl of three years who lived in a low, miasmatic country, but whose family history was not the best; "one paternal aunt was insane, and one cousin a paralytic."

It has been claimed by eminent writers that malaria is not only capable of causing epilepsy, but that the convulsive chills, common to the malady in hot countries, are sometimes substituted for the epileptic paroxysm, just as the psychic epileptic equivalent is believed at times to appear as a substitute for a real epileptic convulsion.

In the second case the subject, a man now thirty-seven years old, stated that an attack occurred when he was a boy of four, and that a maternal uncle was an epileptic.

Marandon de Montyel and Maurice Didé * very thoroughly investigatcd the connection between epilepsy and malaria. In one existing case of epilepsy in which the convulsions had long been in abeyance, the disease became worse when the patient contracted malaria, while others "who had never before had a seizure of any kind became markedly epileptic" under recent malarial infection.

In the latter, however, the author admits the presence of "a distinct family neuropathic proclivity."

In two cases the epileptic attacks came on coincident with the rise of temperature due to the malarial poisoning.

These writers make some observations on the influence of the infectious fevers on epilepsy, which will be referred to at greater length when we come to speak of treatment.

Diphtheria.—Two cases followed this disease; one in a girl eleven years old whose family history contained instances of alcoholism, epilepsy, and rheumatism.   The girl herself was of the lowest possible moral stamina, yet she possessed much intelligence, unimpaired by the disease.   Her attacks occurred once in from three to six months, and have persisted now for ten years.

The second case occurred in a boy of fourteen years, about whose ancestry nothing could be learned.

Measles.—In only one case, that of a boy now fifteen years old, who had the first convulsion just after an attack of measles at six months, was this given as the cause.

Only one out of over 1300 cascs due to this cause seems strikingly low.

Gowers speaks of 12 cases caused by measles, though he fails to mention the total number of cases studied in which the convulsions occurred.

* "Revue de médecine," Dec. 10, 1899.

Meningitis.—Five cases due to meningitis have come under my care.   In two it was confined to the coverings of the brain; in the remaining three it involved the coverings of the cord alone.

In such cases the convulsions appear, as a rule, during the height of the disease, and they are frequently of such severity as to cause cerebral hemorrhages that perpetuate the attacks.

In one case the meningitis came on at the twenty-second year, while in all the rest it appeared before the seventh year.

From all the foregoing, it would appear that any of the specific fevers have the power of inducing epilepsy, first causing reflex or symptomatic convulsions which, in a single attack, or in a longer time in other cases less favorably constituted to acquire the epileptic habit, establish the true disease.

EMOTIONAL SHOCK.

The casual student of the etiology of epilepsy might very properly hesitate to believe that the disease is so often due to the results of emotional shock, as reliable data suffice to show.

Féré says it is beyond doubt that in predisposed subjects a fit may be immediately provoked by a more or less intense shock to the nervous system, and that these physical states are associated with an over-excitability which is very favorable to the evolution of convulsive phenomena.   At the menstrual periods these emotions act even more powerfully by reason of the nervous susceptibility which accompanies this physiologic condition.   The convulsive discharge may occur immediately after the emotion is experienced, but this is not always the case.

The nervous depression succeeding emotional shocks may last some time, and the attack may supervene during the asthenic state, occasionally long after the

initial cause. An epilepsy which manifests itself in connection with some fright may become definitely established and the fits may thereafter appear without apparent exciting cause.

In other instances the attack occurs invariably when the same exciting influence is reapplied; for instance, the sight of a corpse, of a precipice, of blood from an injury, etc.

Of such importance do I feel this too generally unappreciated factor in the causality of epilepsy to be, that I quote the following at length from Sir William R. Gowers on "Epilepsy and Other Convulsive Diseases," 1901, p. 25:

"Of all the immediate causes of epilepsy the most potent are psychical—fright, excitement, anxiety. To these are ascribed more than a third of those in which a definite cause is given. Of the three forms of emotion, fright takes the first place. The relation of this cause to age is, however, very distinct. It is effective chiefly in early life, when emotion is so readily excited, and is most powerful at the transition from childhood to adult life, while after middle life it is almost inactive. Of 173 cases only 14 commenced after thirty years of age, and 145 began under twenty. Of these the majority, 102, began between ten and twenty, only 43 cases before ten. The female sex is notoriously the more emotional, and accordingly the disease results from fright in a larger proportion of women than of men, although the difference is, perhaps, less than might be expected—61 per cent. of women and 39 per cent. of men. It is notorious also that this difference between the sexes increases as life advances. In childhood one sex is almost as emotional as the other, but with puberty men become far less emotional than women. The influence of fright as a cause of epilepsy is in strict harmony with this fact. Under ten years of age the sexes suffer

equally.   Between ten and twenty the male sex suffers less than the female sex, as 3 to 4; between twenty and thirty, as 3 to 13, and over thirty the only cases due to this cause occur in women.

"Predisposition usually exists.   Hence, in speaking of fright as a cause of epilepsy, it must be remembered (as already stated) that its effects are only that of the exciting spark.

"The exact form of fright varies, of course, in different cases, but the list is instructive.   In several cases the cause was some stupid practical joke—a pretended ghost, children shut up in dark cupboards, an alarm of fire, or burglars.   In a few cases the patient had watched other persons in fits.   Alarm during severe thunder-storms was another cause.   One case was that of a soldier who had his first fit a few hours after being terrified, while on sentry duty at night, by the unexpected appearance of some white goats on the top of the adjacent walls of a cemetery, which he mistook for emissaries from the graves."

The foregoing statements of this distinguished physician are impressive.   But I am unable to agree with him in the first statement he makes, namely: "Of all the immediate causes of epilepsy the most potent are psychical—fright, excitement, anxiety."

I feel that they play a large part—one too generally unrecognized—but my experience in over 1300 cases does not lead me to the conclusion reached by Gowers.

This author speaks of 173 cases due (apparently) to this cause, and I assume that they occurred among the 3000 he mentions as being the basis of his studies.   They would thus constitute a little over $5\frac{1}{2}$ per cent. of the total number, a percentage that substantially agrees with that found by us in a close study of 1323 cases.

My point of difference with Gowers lies in the fact

that I found the most potent, immediate, single
cause of epilepsy to be the cerebral palsies of early
life—a distinct physical cause instead of a psychic one,
such as emotional shock, and which Gowers declares
to be the most common.

Referring again to the same series of 1323 cases
I found emotional shock or fright to have been the
cause in 62, or 5½ per cent. Of these 22 were men
in a total of 814, and 40 were women in a total of
509, being about 3 per cent. among the former and
nearly 8 per cent. among the latter, showing the
cause to be nearly three times as active among
women as among men.

This is generally recognized as being the case, and
it is unmistakably due to the less stable nervous system
in women as compared with the opposite sex.

During the first ten years of life, there were 14 cases
due to this cause among 814 men, and 15 among 509
women, which demonstrates the greater susceptibility
of the latter sex even at this early age.

Some writers state there is no difference in the sexes
during the earlier years, but such is not my experience.
During the second ten years only 7 cases occurred
among the same number of men, whereas there were
25 among the women. After twenty there was one
case only in either sex. It seems, therefore, that
emotional shock most often leads to epilepsy in the
female sex, and is most apt to be active about the age
of puberty.

It is also of interest to note the influence of heredity
in such cases at the three age periods named above.
Under ten years, some hereditary influence was pres-
ent in 5 boys and not present in 9. Between ten and
twenty it was present in 2 and not present in 3; while
it was not present in a single case after twenty, thus
making it a factor in 7 cases only out of 22 among
the men. Under ten years it was present in 10 girls

and not present in 4; between ten and twenty it was present in 14 and not present in 11, while it was noted in only one case after twenty (and that at the forty-fifth year), thus making it a factor in 20 cases out of 40 among the women.

The character of the emotional shock or fright is indicated in part in the following: "Fright from seeing an escaped lunatic"; "frightened by a drunken father"; "fright from seeing blood from a cut finger"; "from a dog bite"; "from being chased by a cow."

As an illustration of the type of case in which emotional shock can produce epilepsy, the following is a typical instance:

L. L. had the first convulsion when she was a girl of nine years, due to seeing an escaped lunatic. She has always been "very emotional and easily excited." She had seven or eight attacks at first every twenty-four hours, which condition persisted for some years; then they dropped to four or five a day until 1896, when she was twenty-four years old. At that time they began to appear several days apart. At first they were entirely *petit mal* in character, but later changed to *grand mal* without an aura. She has always fallen in such a way as to injure her face, having several times fractured her nose, and more than once her lower jaw. If there ever was an element of hysteria in her attacks, it disappeared completely years ago, for she is a typical epileptic at this time, bearing in her face the only physical signs pathognomonic of the disease—a mass of scars acquired through repeatedly falling and injuring the same place—a part of the only true *facies epileptica*.

It sometimes happens that when the disease is caused through excitation of the psychic centers, the reapplication of the same form of excitation will bring on an attack. The writer has knowledge of a girl, now fourteen years old, in whom the disease

appeared when she was twelve, and was caused through fright on hearing the fire alarm, and seeing the fire-engines go by one night. While under my care there were two occasions when she heard similar alarms, and each time she had an attack just as she rushed to the window to watch the preparations for putting out the fire.

This is not only reasonable, but logical, as well as psychologic, for the unconscious association of ideas and the results they produce are as definite in action as it is possible for memory associations to ever be.

A gentleman who sat in Ford's Theatre in Washington the night Lincoln was assassinated saw the assassin enter the box in which the President sat; he saw the flash of the pistol and the confusion that followed. In the next row of chairs in front of him sat a lady on whose corsage was pinned a large bunch of heliotrope, a flower of intense perfume. He caught a whiff of the odor of this flower just as he saw the flash from the pistol and heard the noise, and for thirty years after, when he told me the story, he never smelled the odor of heliotrope without seeing the whole tragic scene reenacted just as it occurred before him that night.

So it is that the sensory stimulation of any center or faculty that is organized into a definite action may be produced indefinitely under the reapplication of the same or similar causes.

"There is," says Thomas Watson in his classic work on "The Practice of Physic," "yet another very singular occasional cause of epilepsy that deserves to be mentioned, namely, the sight of a person in a fit of that disease."

This distinguished writer states that this fact has been noted time and again; that patients who already have the malady will fall in a convulsion at the sight of another one; that those not previously

affected may acquire the disease in this way; and even that the disease will "now and then run through a boarding-school or through the ward of a hospital." He also cites the case of a young man, employed in the care of an epileptic suffering frequent and violent attacks, who himself became a victim of the disease at the end of seven weeks.

Watson speaks of the possibility of these attacks being due to imitation, or to feigning, or most likely to hysteria, but finally decides they are epileptic.

I dissent from the view of this writer that genuine epilepsy may become epidemic in schools and in hospitals. In my opinion the instances of this kind should unquestionably be classed as convulsions due to hysteria.

I agree with him that it was possible for the attendant to acquire epilepsy after seven weeks of arduous labor in the care of a violent epileptic; not, however, so much because his charge had epilepsy, but because of the great stress, mental and physical, and the extreme fatigue such labor imposed on him, combined to some extent with the possibilities of suggestion on an individual so prepared.

As to the power of an epileptic attack to excite a seizure in another epileptic, I do not for a moment doubt, though it is comparatively rare, and I believe the constant association of epileptics, sensitive at first in this way, with other epileptics who have frequent seizures, causes them in time to lose such sensibility entirely.

I have observed that such instances in a large epileptic community become in time extremely infrequent. A few days after a large number of epileptics of all types and characters had been brought together, about 75 of them had to eat in a common dining-room. A violent seizure occurred at the dinner-table one day; the table was knocked over, and

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great noise and confusion resulted.   In a few seconds
four other epileptics were in convulsions, as the result
of the first seizure.

In a few days the occurrence of a single fit in this
room no longer had the power of causing attacks in
others accustomed to the disturbance.

A chief nurse reported an instance in which a man
had a violent attack at midnight and awakened his
companions about him, three of whom at once had
epileptic convulsions; but they were not hysterical
in any degree, the shock of sudden fright having
brought on the attacks.

I recall two instances in which a young woman
went into a convulsion at the sight of a companion so
affected, both having occurred at night.

It has also been observed that when epileptic men
and women meet in common assembly for religious
worship, or to see a play, both sexes have more at-
tacks proportionately than when one or the other
attends alone.   The reasons for this may be found in
the psychic stimulation of the sexual passions, a
recognized exiciting cause in some cases.

According to Masso, Pinel always began the exam-
ination of a patient by asking him whether he had
not had some fright or great vexation.   The same
author states that through the vivid impressions
emotions may produce, the same effects may be sus-
tained, as, for instance, through a blow on the head or
some physical shock.   Fear causes the loss of conscious-
ness, sight, and speech.

Beger reports the cases of two perfectly healthy old
men, one sixty-five, the other seventy years of age,
who had epileptic fits immediately after being fright-
ened, though they had never suffered in that way
before, nor were they predisposed to such a disease.

Kohts, in his account of the maladies caused by
fright during the seige of Strasburg in 1870, gives a

minute description of the cases of paralysis agitans and of convulsions which he observed; while we find numerous references in Hack Tuke's " Dictionary of Psychological Medicine " to the emotional state as a cause of epileptic attacks, in many of which were characteristic convulsions.

In Hecker's admirable work on " The Epidemics of the Middle Ages," mentioned by Tuke, there is a description of the "dancing mania" and "tarantism"; parts of which are of interest in this connection. "It was," says Hecker, "a convulsion which infuriated the human frame in the most extraordinary manner and excited the astonishment of contemporaries for more than two centuries. As early as 1374, assemblages of men and women who had come from Germany, were seen at Aix-la-Chapelle, and, united by one common delusion, they exhibited to the public, both in the streets and in churches, the following strange spectacle: They formed circles, hand in hand, and, appearing to have lost control over all their senses, continued dancing for hours in wild delirium, until they fell to the ground in exhaustion. While dancing they neither saw nor heard, but were haunted by visions, their fancies conjuring up spirits whose names they shrieked out. When the disease was completely developed the attack began with epileptic convulsions."

There is probably no cause to doubt that these frenzied states were at first free from any taint of real epilepsy, while there is as little to doubt that the long continuance of such states may lie at the root of true epileptic convulsions. We need only remember that a single convulsion, by leaving what may be termed a physical stain, definitely thereafter perpetuates its kind.

Prolonged Anxiety, Grief, and Over-work.—Like insanity, no list of causes of epilepsy would be com-

plete which did not include forms of mental stress or disorder, acute or chronic, due to great anxiety, over-work, or grief. It has been my experience that such causes, comparatively rare at best, exist in far greater proportion in women than in men, and that they are most active about the age of puberty.

On this point I do not agree with Sir William R. Gowers, who states that in a series of forty-eight cases apparently due to this cause, males predominated, and that "this cause is effective late in life more frequently than most others."

It has also been my experience that over-work and great anxiety simply act as immediate or exciting causes in cases that bear a predisposition to the disease, this predisposition being effectively increased by the epoch of puberty.

Considering all these factors together, I found them to be the cause of epilepsy in 16 out of 509 women, and 6 out of 814 men, being about 3 per cent. of the former and about two-thirds of 1 per cent. of the latter.

Several cases among the women appeared in girls at school, ranging in age from twelve to sixteen, the "stress of ambition" being the exciting cause in some of them; the striving for school honors; long hours of study in vitiated atmospheres; improper food; inattention to matters of health, and things of like nature common to the age of school-girl life.

I recall particularly the case of a girl of excellent natural ability, bright, quick, energetic, and especially devoted to music. She was pushed along in her work by a no less ambitious but most unwise mother, with the result that at the age of fourteen years the girl developed epilepsy in the worst form, and at the age of eighteen was hopelessly demented. She is still subject to frequent convulsions.

CHAPTER V.

ETIOLOGY.—PART II.

Trauma. Proportion of Cases due to Trauma. Gastrointestinal Dis-
orders. Lead Poisoning. Renal Disease. Heart Disease. Tobacco.
Disorders of Menstruation. Pregnancy and Maternity. Masturbation.
Syphilis. The Influence of Sleep. Sporadic Causes: Congenital Brain
Defects; Hypertrophy; Hydrocephalus; Fatigue.

TRAUMA.

THE chief point of interest in connection with the
surgical treatment of epilepsy, which is fully discussed
in a separate chapter, is based almost wholly on the
fact that injuries of certain kinds about the head and
in connection with the spinal column, not infrequently
lead to epilepsy.

It is also claimed that, in addition to injuries of the
cerebrospinal nervous system, injuries to peripheral
nerves may cause epileptic phenomena, and, while
I agree with the possibility of this, such a case has
not come under my observation that I recall.

It may be noticed that I speak of "epileptic
phenomena" due to such causes instead of epilepsy
proper, for I do not hold that such convulsions are
anything else than epileptiform—like epilepsy—in
the beginning.

It was by irritating or cutting the sciatic nerve that
Brown-Séquard succeeded in rendering guinea-pigs
epileptic.

Traumatic lesions of the cranium and the cerebrum
cause epilepsy without any predisposition to the dis-
ease. In such cases there is generally some physical
evidence of the injury, or a clear history that such an
injury was received, though there are cases in which

convulsions follow simple concussion independent of any actual physical lesion whatever.

A child three years old fell from a veranda to the ground, a distance of six feet, striking on its head. It remained unconscious half an hour, then a general twitching of the body occurred, which was shortly followed by a severe general convulsion, and fifteen minutes later by a second one much less severe, after which vomiting set in and consciousness was soon restored.

Here the convulsions were due to the concussion, but had there been intracranial injury of any kind, like hemorrhage, permanent organic epilepsy might have resulted.

Westphal succeeded, according to Féré, in rendering guinea-pigs epileptic through concussion by striking them repeatedly on the head.

A blow on the head which fractures the skull or causes intracranial hemorrhage may not be followed by convulsions for some hours, or for several days, or until a considerable local effusion has taken place; the irritation resulting from the effusion causes the convulsions—a fact often proved by the cessation of the seizures on the removal of the clot, an illustration of which is found in the case of the midshipman at Annapolis, reported by Keen.*

Gunshot wounds in which the bullet lodges in the brain and is not removed, may cause convulsions likely to change into epilepsy. The writer recalls two cases of this kind, in one of which the bullet was photographed *in situ* (by means of the *x*-ray), but it could not be removed, as it was at the base of the brain in an inoperable position.

It is a distinction of these cases worth mentioning now, that the mental condition rarely deteriorates until the disease has existed for years, and not even then

* "The Philadelphia Medical Journal," Dec. 13, 1902.

Plate 7.

Fig. 1.   Fig. 2   Fig. 3.

Fig. 4.   Fig. 5.   Fig. 6.

Figs. 1 to 6 show the order of progression of attacks in a case in which the initial spasm—519 times in succession in forty-nine hours—was confined to the first phalanx of the right thumb. Death from status epilepticus followed at the end of that time. Careful pathologic examinations of the right thumb and hand centers in the brain failed to show any lesion other than a condition of marked cell vacuolation such as would be expected to follow extreme fatigue. Exhaustion paralysis often follows localized cortical "discharges" in epilepsy of this kind.

unless the original stamina of the patient was poor and the attacks frequent and severe.

Roswell Park* relates the case of a man who was wounded during the charge up San Juan Hill, by a bullet which ploughed its way over the skull and injured the brain, so that three years later the man had hemiplegia, and epileptic seizures which appeared weekly.

As a rule, the convulsions due to trauma are of the Jacksonian type, which appear locally in one arm, or leg, or on one side of the body. This is because the injury is local, affecting, as a rule, a limited portion of the cortex, generally in or near the motor region.

Such cases, as I have before stated, are referred to as organic epilepsy, and even though they begin locally, they occasionally show a tendency to spread and involve all parts of the brain, and necessarily all parts of the body as well.

We must not, however, be led into supposing that every epileptic seizure that begins locally is Jacksonian and due to a fixed organic lesion capable of demonstration.

I reported the case of a woman† who had 519 well-marked attacks in forty-nine hours, the initial manifestation of each attack being the quick, sharp contraction of the first phalanx of the right thumb, then the whole thumb, next the hand, the forearm, the whole arm, and finally the right side of the body. She died in status epilepticus which supervened after she had had about 200 attacks. A careful microscopic study of numerous sections of the brain from the right thumb center and its vicinity showed no lesion whatever beyond a condition of cell-vacuolation, that we would naturally expect as the result of extreme exhaustion. (See Plate 7.)

It is of interest to add that, while the seizures in this

* "American Medicine," Nov. 22, 1902.
† "New York Medical Journal," March 13, 1899.

case invariably began in the right thumb, they quickly
spread to the entire right side of the body, and re-
mained there as long as the patient was kept pro-
foundly under the influence of chloroform, which was
done during nine of the forty-nine hours she was ill;
but when the chloroform was withdrawn, they quickly
involved the entire left side as well.

The same manner of extension of the attack may
take place in cases in which there is a definite, circum-
scribed, organic lesion, and in the surgical treatment
of such cases it is possible for the operator to be misled
into cutting down on a point to which the symptoms
have radiated, and which does not represent the true
seat of the lesion.

In the milder cases of Jacksonian epilepsy, as I
have before pointed out, consciousness is not lost, in
some cases not even greatly impaired, the patient
being able to recall after the attack what took place
while it was in progress. (*Vide* case J. P., described
under Jacksonian Epilepsy, Chapter V.)

Some writers claim that lesions of the spinal cord
may produce epilepsy, and more often epileptiform
manifestations.

Brown-Séquard observed the occurrence of epilepsy
after experimental lesions on the spinal cord of guinea-
pigs, such as complete transverse sections of the pos-
terior column, of the posterior horns, and of the anterior
and lateral columns, and Féré states that in man a
number of cases have been observed in which epilepsy
resulted from compression of the cord by a tumor,
traumatism, or some disease of the vertebræ.

A letter-carrier, thirty-four years of age, suffering
from multiple sclerosis of the brain and spinal cord,
developed well-marked convulsions which appeared
at first once a month; later they increased to three
or four a month, then occurred daily. Suddenly
they ceased for almost a year, and during that time

his malady progressed rapidly, the pathologic changes in the cord having become so far advanced as to entirely destroy control of the centers governing the bladder and the rectum.

The convulsions in this case were thought at first by the attending physician to be due to a "tumor in the brain," and the patient was trephined, though necessarily without beneficial results.

Five years after the beginning of his illness, his convulsive attacks appeared to cease entirely, while the multiple sclerosis grew steadily worse.

In the writer's opinion, such cases should not be classed as genuine epilepsy, for it is clear that the convulsions were merely an incident of a graver organic disorder, just as they might have been of some simpler organic disease severe enough only to excite reflex convulsions.

It is interesting to note in this connection the functional division of the cerebro-nervous system into three parts by Hughlings-Jackson, if for no other purpose than that of using such divisions as aids in explaining the probable seat of the lesion in epileptic seizures of different types. Jackson divides the nervous system into three levels: *Highest, middle*, and *lowest;* the latter corresponding practically with Marshall Hall's *spinal system* in including the gray matter of the spinal cord in its upward prolongation in the brain as far as the oculomotor nucleus.

The centers in this "level" are sensorimotor and govern the fixed automatic acts like walking, and also control the centers of respiration, deglutition, defecation, and urination; and· we may say these centers are removed *one* degree from the periphery.

Above this spinal system, or *lowest level*, lie the centers of the *middle level*, "functionally definite, but anatomically not so readily definable" as were the lowest centers.

This level, too, is sensorimotor in character, being on the motor side and practically synonymous with the motor area outlined by Ferrier, including the corpora striata. Sensorily speaking, this middle level includes probably the greater part of the temporo-sphenoidal lobe, the gyrus fornicatus, and the inferior parietal lobule.

The centers of the middle level are far more delicate and complicated and far less automatic than those of the lowest level, and we may say they stand *two* degrees removed from the periphery.

The third and *highest level* embraces the superior intellectual centers in the brain; those that are believed to lie in the anterior or preparietal lobe and in the posterior or occipital region. These are still less automatic than the centers of the middle level, and they in turn are, so to speak, *three* degrees removed from the periphery.

It is still a mooted question as to whether the frontal lobes are motor to any extent or not. Hughlings-Jackson maintains they are, and we are justified, for the present at least, in assuming all parts of this *highest level* to be sensorimotor; the sensory functions vastly predominating in importance over the motor.

In this level we must undoubtedly look for the lesions that underlie the psychic or purely mental forms of epilepsy—the silent seizures of the mind, the "blanks," "absences," "darknesses," spoken of in Chapter I.

This general mapping of the cerebrospinal nervous system into three parts, where each part represents the basis from which epileptic convulsions of different types spring, is of practical interest in a diagnostic way, even though the functional and anatomical relationship of the three levels generally acts in harmony in the closest alliance and as a correlated whole.

Still there are numerous instances, as we shall see later on, where each acts independently of the other, and so it is they afford a basis for the explanation of certain circumscribed fits. In this connection I will only add that convulsions due to spinal cord diseases, pure and simple, may, not infrequently, fail to *reach up* into true epilepsy at all, because the sudden excessive discharge or *explosion* of nervous force in such comparatively low centers as those in the cord fails to seriously involve any part of the brain.

PROPORTION OF CASES DUE TO TRAUMA.

Trauma is more frequently the cause of epilepsy in men than in women. In 509 women who came under my observation, it was the cause in 18 only, or about 3½ per cent.; while in 814 men it was the cause in 70, or about 8½ per cent., being nearly three times greater in men than in women.

In 73 cases out of 88 occurring in both sexes, the injury was received before the twentieth year, again showing it to be the young who suffer most, other age periods being as follows: Boys under ten years, 31 cases; from ten to twenty years, 25 cases; over twenty years, 14 cases. Girls under ten years, 11 cases; ten to twenty years, 6; over twenty years, 1 only.

Among the ways in which injuries that lead to epilepsy are received, may be mentioned the following from personal observation: " Kicked on the head by a horse," "fell on the ice while skating," "struck on the head by a falling limb," "fell from a scaffold," etc.; while the greater number are simply ascribed to an "injury to the head," most of these being received by the young, whose fear of the liability to injury is over-balanced by a lack of judgment and a healthy excess of animal life that leads them into perilous positions.

In other rare cases epilepsy may directly follow injury to the spine.

When C. M. H. was eight years old, he was hurt by a man kicking him "over the lower end of the spine." From that time until he was eleven he had spells of "losing himself," which came on from three to four times a day. Then he began to have *grand mal* seizures which were announced some time before they appeared by a "general chilly sensation."

The following is illustrative of the worst form of epilepsy due to trauma:

W. H. J. first came under my care in 1896, when he was twenty-three years of age, having been an epileptic five years. His family history was good. As a boy he worked on a farm; he was never dissipated and never ill until his epilepsy began in his eighteenth year.

In passing along the road during a wind storm, he was struck on the right side of the head just above the ear by a falling limb. He remained unconscious for some hours; there was a little fever, and he stayed in bed for several days.

The attending physician could not find any evidence of fracture of the skull, nor were there any symptoms of paralysis or of any disturbance of the brain until he developed convulsions "a few weeks later." These were considered the result of the blow on the head, though for some reason an operation was not undertaken.

The convulsions were all of the *grand mal* type and most severe. There was no definite record of the frequency of their occurrence until he was admitted to the Craig Colony five years after the receipt of the injury, at which time they occurred on an average of one every two weeks, being both nocturnal and diurnal.

During the inter-paroxysmal periods he was fairly intelligent, though his mental processes were painfully slow and at times extremely difficult. He soon began to have "periods of marked somnolence and stupor" that came on at times wholly independent of any

visible seizure, though it cannot be said that he did not have psychic seizures before them which escaped notice, while at other times, such periods followed single attacks or a series of them, the exhaustion which followed the latter being most profound.

When passing through the worst of these periods, he was incapable of being aroused; the plantar and corneal reflexes were abolished, the pulse-beat full and slow, and respiration was stertorous and labored.

Trephining was considered, but was abandoned because of the length of time that had passed since the receipt of the injury, and because of the generally unsatisfactory condition of the patient.

He was given the bromids in small doses, thinking they might check the seizures, but they "depressed him mentally and physically" to such a degree that they had to be withdrawn, while they wholly failed at any time to lessen the convulsions either in frequency or severity.

At one time he averaged three seizures a week for six months, while at another time he went six months without any at all. The attacks of stupor increased in frequency during the last two years and he passed ultimately into status epilepticus as the result of a long series of attacks. His temperature went up to 104° F., and death followed from exhaustion.

The autopsy showed no evidence of a former fracture, though the skull-cap was much thickened anteriorly. The dura was adherent at the site of the Pacchionian bodies on the sides, causing laceration of the brain and its covering when it was removed. A marked atrophy of the central portion of the right temporosphenoidal lobe was present, as well as atrophy of the right posterior parietal lobe involving the motor region. The pia was firmly adherent to the brain over the atrophied region. Small cysts were found in the choroid in the lateral ventricles, the cyst on the right side being the larger.

The convulsions in this case were clearly due to the results of the injury, these results being in the form of a subdural hemorrhage that compressed the brain in such a manner as to interfere with its nutrition, with

consequent atrophy, the adhesions being the result of the local inflammatory changes caused by the blow.

GASTROINTESTINAL DISORDERS.

The epilepsies due to derangements of this part of the nutritional system are, in my opinion, of greater importance than they are generally supposed to be.

It is less difficult now to give such derangements a reasonable clinical value than it is to elaborate the method of their action in the laboratory or otherwise. However, if the former is true, the latter in time must surely follow, and we have full confidence that the chemical pathology of the future is destined to play a rôle of great importance in isolating the cause of the disease in some cases in which it is yet obscure as a laboratory problem, and as yet not fully proved in a clinical way; only based on clinical evidence that seems fairly conclusive and certainly of much practical importance.

By gastrointestinal disorders I do not mean so much to include diseases of the alimentary tract, like chronic diarrhea and ulcers of the stomach, as I do acute indigestion and intestinal toxemias.

Irrespective of the absorption of poisonous matter into the blood from the alimentary canal, the irritation of the peripheral nerves (visceral) in this locality may alone cause reflex convulsive phenomena that may persist as epilepsy; and it is a notable fact that the irritation of such nerves seldom causes sensations which reach consciousness, while any irritation of the cerebrospinal nerves is at once realized through the pain induced.

Beginning with the buccal cavity, we find that dental caries has been ascribed as a cause of the disease in some cases, though we incline to doubt that this alone is ever capable of producing it. It is far more probable that the caries is associated with some

disturbance of the alimentary tract which is the actual cause, though we should never fail to carefully examine the mouth and teeth of every epileptic and correct all possible sources of irritation in either, remembering that the important process of nutrition begins here, and that the best digestion is imperatively demanded by the epileptic.

In many cases in which difficult dentition is supposed to be the exciting cause of epileptic phenomena, it is always worth our while to look carefully for evidence of gastric or intestinal derangement.

In speaking previously of the effects of difficult dentition, it was noted that *indigestion* or *stomach trouble* was often coupled with it, and I do not doubt that in some of these cases they constitute the sole cause, the eruption of the teeth having nothing to do with it.

A rather broad inference of a practical character may be made in this connection to show the manner in which the products of intestinal indigestion cause convulsive phenomena, and later on true epilepsy.

I have previously referred to "the storing of nervous force or energy and the manner of its discharge." "If," as Gowers says, "we can perceive the conditions that underlie normal nervous action in the nerve centers, the phenomena of epilepsy will become in some degree less mysterious. In health, energy is liberated in instant response to a definite stimulation. Such capacity for instant action involves a delicate equipoise of the processes for the liberation of nerve force and for its restraint and control. The *balance must depend on the processes of nutrition in the nerve structures*, for the liberation of energy depends on the occurrence of chemical processes under the influences of life—processes which must ever be on the verge of destruction."

Properly nourished and under proper inhibitory con-

trol, sensory and motor nerve cells remain, so to speak, in *statu quo*, awaiting the stimulus that will cause them to give off their energy in a normal way. In such cells the equilibrium is justly maintained. In the epileptic state this equilibrium is temporarily impaired or destroyed. The cell may be undersized, or poorly insulated, and incapable in the first place of storing a normal amount or degree of energy, and in the second place of retaining it after it is stored—holding it, we may say, against calls abnormal in kind.

There is undue readiness of the cells to act, to respond to vagrant and unphysiologic forms of stimulation, and so it is that dissipated, purposeless movements, convulsions, or even a series of them, is the result.

Now note the one factor that seems to be capable above all others of disturbing this marvelous balance, that wonderful state of cell-equilibrium, namely, the little-understood processes of nutrition, whether in health or disease.

Nutrition can be disturbed in other ways than merely through the media that carry pabulum to the cell, for we have seen how emotional shock may so affect the function of nerve tissues as to disarrange its balance, causing at first irregular nerve discharges, and later on, epilepsy; but it is the character of the material carried to the cell for the purpose of its constant reconstruction that has most to do with its type of action.

If the stomach and intestinal canal do their parts in a healthy, natural way; if they digest, absorb, and create only the pabulum the body needs for its healthy preservation, there need be no convulsions due to faults in the action of this part of the human economy; but the stomach and intestinal canal are palpably at fault at times, for they manufacture a variety of more or less harmful substances, some of them distinctly poisonous.

According to Bouchard,* the apparently most inno-
cent foods, including the flesh of muscle, are toxic at
times, because of the mineral matter, chiefly potash,
they contain.

Bile, through its coloring matters, may also become
poisonous. It is claimed that the poison in an extract
of 2.5 grammes of putrified meat is sufficient to kill.
The toxic effects of constipation are varied and well
known, being due chiefly to potash and ammonium,
and the combination of organic principles which in-
cludes alkaloid substances.

The chief subjective symptoms of putrefactive
changes going on in the intestinal canal consist of
fatigue, depression, headache, buzzing in the ears,
visual disturbances, vertigo, and irritability of temper
which may often be marked.

Bouchard speaks of the possibility of convulsions
in cases of dyspepsia in which there is urea of sufficient
degree to cause nausea and vomiting, and this, too,
without any disease of the kidneys, only an incapacity
on their part to help eliminate the products of fer-
mentative putrefaction going on in the alimentary
canal. According to modern chemical pathology,
there is scarcely an adjunct to the entire nutritional
system, including all its anatomical divisions, and their
several secretions, that does not suffer serious functional
impairment or disease, capable of hindering or de-
stroying nutrition, and we should seek to ascertain
the possible existence of any of these in every case that
comes under our care.†

Finally, individual susceptibility to certain foods,
harmless in most persons but poisonous in others,
should not be overlooked; such foods usually consist

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* "Auto-intoxication in Disease," 1895.

† The author commends most highly the reading in this connection of
Herter's book on "Chemical Pathology," especially the chapter on the
Chemical Pathology of Intestinal Indigestion.

of shellfish and certain fruits, including peaches and strawberries.

MISCELLANEOUS CAUSES.

Lead Poisoning.—Most authorities agree that epilepsy may occasionally be due to chronic poisoning from lead, constituting *saturnine epilepsy*, though I believe such a cause is extremely rare, for in a total of nearly 1600 cases it was the cause in one only.

A boy of four years scraped a quantity of fresh paint from the side of a house and ate it, developing shortly afterward violent convulsions which lasted, in a diminishing way, for several days, when they ceased altogether. He then remained free from them until the age of fourteen, when the stress of puberty, combined with the effects of masturbation, caused them to recur and become permanently established.

According to Féré, who mentions Tanquel des Planches, Leuret, Grisolle, and others in this connection, plumbism forms a favorable field for the development of epileptic manifestations, intoxication from it producing acute epilepsy which may run into serial attacks that end in status epilepticus or in delirium.

I do not doubt the power of this agency to excite toxic convulsive phenomena, though the comparative rarity of such intoxication makes epilepsy from it infrequent.

Tobacco.—In six cases that I have seen, all being boys under sixteen years of age, *cigarette smoking* was believed to be the exciting cause of the disease. It is worth noting, however, that it appeared in all of them about the age of puberty, and mostly in boys whose ancestry was not good, which two important facts, together with the tendency to masturbation at this age, should cause us to be careful in ascribing the cause to cigarette smoking alone.

I do not doubt the power of intense poisoning from nicotin to undermine the stamina in selected cases in a way to make the individual more susceptible to disease, among these being epilepsy and insanity.

An adult male epileptic declared that the chewing of tobacco always made him worse, and resolved time and again to discontinue the practice, but could not do so.

Besides the possible poisonous effects of the tobacco in such a case, the gastric disturbance, the toxemia, induced by it may have had something to do with bringing on the convulsions.

Gowers speaks of a boy whose epilepsy came on as the apparent result of frequent nausea caused by working in a tobacco factory.

A woman of twenty-eight, whose ancestry was bad— epilepsy, alcoholism, and tuberculosis being in the family, in whom the disease came on at seventeen years, after scarlet fever—claimed for a long time that she never had a convulsion when she smoked, though the observations of her physician did not bear this out. Her fondness for the habit probably caused her to make this statement because she did not want to give it up.

Renal Disease.—We have previously referred to scarlatinal nephritis as a cause of epilepsy, noting its presence in 3 per cent. or more of all cases, and the forms of renal diseases referred to here more particularly include the varieties of Bright's disease.

The neurologist does not come into contact with the convulsive disorders due to uremic poisoning as often as the general practitioner, and the latter is not inclined to treat such phenomena as epileptic.

The influence of the kidney when diseased in producing morbid mental states has long been recognized; much valuable scientific work in this connection having been done by Prout and others, while the subject has been very carefully studied clinically.

On the other hand, the convulsive phenomena due to kidney lesions have also long been recognized, though not systematically studied.

Convulsive attacks in uremic poisoning may develop with a warning or be preceded by such symptoms as pain in the head, restlessness, and malaise; and, according to Osler,* such convulsions may be general and identical in character with those of genuine epilepsy, minus the epileptic cry.

They may occur in rapid succession, the patient being unconscious during the inter-paroxysmal period. Usually the temperature is below normal, though it may occasionally be above.

Jacksonian epilepsy may occur in uremia, while uremic amaurosis (blindness) may appear and persist several days. Hearing may also be temporarily lost.

When the convulsions of uremia are followed by hemiplegia, or monoplegia, established organic epilepsy may be the result. The apparent rarity of epilepsy due to the results of grave kidney diseases in old persons is due to the great fatality of the affection, the patient generally succumbing before epilepsy has become fully established as an independent affection.

Disorders of Menstruation.—These may be either functional or organic, and almost any pathologic cause that deals with the period of life, in man or woman, covered by the epoch of puberty, from the twelfth to the sixteenth or seventeenth year, is capable of misinterpretation, for the diseases that come to light at this period are so numerous that it is not difficult to lay more importance on one supposed causative factor than that factor deserves.

Substantially all authorities agree that irregularities in the menstrual function may produce a long line of neurotic manifestations, prominent among them being epilepsy.

* "Practice of Medicine," 1895.

It has long been a semi-medical tradition, and indeed it is still in some localities, that the establishment of the menstrual flow is apt to cure or favorably influence ills of a nervous order suffered for years up to that time; but there is an abundance of clinical observation now to show that in the great majority of cases the opposite is more likely to be the result, especially in epilepsy.

Voisin, Féré, and Gowers agree that the menstrual function coincides with an exacerbation of the fits in this disease, and progresses thenceforth in a markedly periodic form. This is true to a large extent in many cases. I have witnessed it in a score or more in the hundreds of epileptic women who have·come under my care.

Voisin goes so far as to say that menstruation is "the signal of explosion of epileptic attacks. In our service I find a quarter of the patients at least in whom menstruation has this influence."

Once established, the attacks may become aggravated by the onset of dysmenorrhea or suppression of the menses. Women who have epilepsy from such causes will in most cases be found to possess a neurotic stamina, though some may become epileptic solely through the stress of irregularities of the reproductive organs, and it is often a matter of importance to determine how far functional disturbances are responsible and how far they are organic.

Somers treated a patient by perforation in a case of epilepsy in which the cause was an imperforate hymen that prevented the evacuation of the flow, after which the convulsions ceased. Such a cause is not uncommon, many cases of the kind doubtless occurring, that are not reported. The retained flow becomes septic, and poisons the nervous system by absorption.

A somewhat similar case (elsewhere mentioned)

came under my own observation. A married woman, thirty-seven years old, had an impervious uterus and had never menstruated. The organ was substantially a cyst, and vicarious menstruation appeared each month in the groin.

The convulsions that developed at puberty had persisted ever since, and became especially prominent about the time of the monthly flow.

For several reasons it was deemed best to remove the uterus with all its appendages, after which the attacks ceased entirely, and three years later they had not returned.

The Menopause.—The menopause is a very infrequent cause of epilepsy, or even of reflex epileptic phenomena. Up to this time, I have seen but two cases due to stress incident to the termination of menstrual life. The reasons for this are twofold, as follows: 1. Epilepsy is essentially a disease of early life. 2. A woman who has successfully run the gauntlet of the epochs of dentition, of puberty, and maternity need have little to fear from that of the menopause when it follows anything like a normal course in its establishment.

Pregnancy and Maternity.—It is far more common to see epilepsy arise during pregnancy, or immediately after delivery, than at the menopause. Gowers speaks of ten cases in which the first fit occurred without obvious cause during pregnancy, and of five in which it occurred after parturition. Three cases which occurred as the result of maternity have come under the writer's observation.

As a rule, such cases at first are described as eclamptic, and the great majority of them are checked before they pass into epilepsy; while others soon acquire all the essentials of the true disease. Many of them under modern medical and surgical procedures are preventable, and we mention some instances to show the

disastrous effects that may follow failure to apply early and correct treatment:

L. M. came under my care when she was thirty-nine years old, having had epilepsy since she was twenty-three, when the first convulsion appeared soon after the birth of her child. She never had a convulsion prior to that time. During the first five years the attacks, very frequent at first, gradually grew less so, but more severe in kind, alternating between *grand mal*, *petit mal*, and psychic. Gynecological examination showed a much inflamed cervix uteri and marked vaginal cystocele, both doubtless of very long standing.

The patient's mental condition was markedly impaired, epileptic dementia being well established, while the attacks still occurred from four to five times a month.

It cannot be reasonably doubted that epilepsy in this case was due to some injury suffered at the time of parturition, the prompt repair of which might have saved the woman from epilepsy and insanity in the end.

M. McC. had the first convulsion at thirty-three years, immediately after childbirth, and when we saw her at the age of thirty-seven, she was a confirmed epileptic, having several attacks each month, all of the *grand mal* type. Her mental faculties were largely destroyed, and she was subject to maniacal outbursts of great violence, having at such times numerous unsystematized delusions and hallucinations. She finally became continuously insane and a subject for commitment to a hospital for that class.

M. H., a married woman of twenty-nine when I first saw her, developed epilepsy at the age of twenty-one, when she had a miscarriage, this being her second pregnancy; the first had required instrumental delivery, harmless alike to mother and child.

The miscarriage was induced at the sixth month because it was deemed necessary to save the woman's life which was threatened by the frequency and violence of the eclamptic attacks. Shortly after these had subsided, she had slight psychic seizures that persisted occasionally for about four years, when

grand mal attacks began to appear. After the lighter
attacks she was frequently automatic, going about
in a dazed, subconscious state, doing a number of
little things that she had no knowledge of, and it was
not long before evidences of epileptic dementia ap-
peared, which was soon fully established. Prevention
in this case, or the possibility of early cure, was not
to be so much expected.

Grandin and Jarman* state that eclampsia occurs
once in from five to six hundred pregnancies, unless
the woman develops an acute nephritis during preg-
nancy, when the proportion arises to about 25 per
cent. In this latter class of cases the maternal mor-
tality averages about 30 per cent.

As many as from seventy-five to one hundred
eclamptic attacks may occur in twenty-four hours,
and whatever else the prime etiologic factor in such
cases, a secondary factor is uremia, this being assured
through good results attained in eliminating the full
amount of urine.

Hirst ("Textbook on Obstetrics," 1899), in review-
ing the nervous complications of pregnancy, says:
"Epilepsy is a rare complication of pregnancy and,
as a rule, does not influence unfavorably the course
of gestation. The convulsions are often absent during
pregnancy, but make their appearance again during
and after the puerperium, or upon the reappearance
of menstruation after the child is weaned."

Among the causes of epilepsy at this period we
must not overlook phlegmasia alba dolens, or milk
leg. The clot in such cases may be carried to the
brain, one instance of the kind being known to the
writer, or the general septicemic state may directly
poison the cerebrum, or the local inflammation itself
may cause reflex convulsions.

Epilepsy due to pregnancy or maternity is apt to

* "Pregnancy, Labor, and other Puerperal States," 1896.

assume the gravest aspect, and when eclamptic attacks appear they ought to be vigorously treated with a view to preventing as soon as possible the *habituation of the epileptic impulse.*

Masturbation.—It is exceedingly difficult to arrive at any satisfactory estimate as to the number of cases of epilepsy due to this cause. The habit is common among epileptic boys, and it is often impossible to say whether it was acquired before the epilepsy or after.

Gowers contends that it can be established as a cause only in cases in which circumcision checks the convulsions, and that true epilepsy is seldom caused by it, though it may be a frequent cause of severe *habit spasms* in boyhood. It seems to me that this aims to make a distinction without a difference.

Habit spasms in such individuals, at this age, and due to this cause, which are general in character, mean, in my opinion, nothing more or less than habit epilepsy.

I have personal knowledge of nine cases in boys ranging in age from twelve to seventeen years, in which the epilepsy was due to this pernicious practice, while it was the cause in the case of one girl.

Dana says that masturbation is an occasional cause of epilepsy; Holt classes it among the reflex causes of the disease; Lauret put it down as the determining cause in 12 out of 66 cases that he studied; while Nothnagel refers to it in this way: "Hereditary epilepsy is generally esteemed incurable, yet Herpin and Reynolds, for example, have seen marked exceptions to this rule. The same holds, according to the latter observer, for the disease when due to masturbation."

Voisin writes at some length on the abuse of coitus as a determining cause of epilepsy, adding: "Masturbation plays the same rôle in the production of epilepsy as abuses in coitus."

Syphilis.—Infection due to the poison of syphilis

seldom causes epileptic manifestations that rise to the
true form of the disease, unless the brain or its men-
inges are involved, and even then the convulsive
attacks are more apt to simulate Jacksonian epilepsy,
involving one leg or arm, or a part of the body only.

A large number of such cases have come under my
care. The "spinal epilepsy" described by Brown-
Séquard as mentioned by Gowers,* and which has a
superficial resemblance only to true epilepsy, may
possibly be due to syphilitic involvement of the spinal
cord, though more often to other degenerative pro-
cesses.

Every practitioner's personal experience affords
abundant confirmation of the fact that brain syphilis
causes a type of convulsions which not infrequently
runs into genuine epilepsy.

The character and course of this type of the disease
are quite distinctive, closely resembling the epilepti-
form convulsions of paresis in many cases. Physi-
ologic and pathologic researches during the past
quarter of the century have shown that definite lesions
of definite portions of the brain give rise to certain
fixed symptoms. We know that the motor tracks
take their origin from the central convolutions, and
as these tracks become smaller they pass through the
corona radiata, the internal capsule, and the cerebral
peduncle and pons to the crossing of the pyramids.

It is clear, therefore, that a small lesion at the cortex
will produce a circumscribed paralysis of a paretic con-
dition, while a lesion of similar size in the internal
capsule, where the fibers coming from the cortex are
crowded into a small space, may produce a complete
hemiplegia.

Monoplegias are frequent symptoms of cortical
lesions, and can with certainty be referred to this
locality when symptoms of irritation (convulsions)

* "Diseases of the Nervous System," Vol. I, 1899, p. 265.

appear in the affected parts. Numerous scientific teachings show that such centers are frequently the seat of syphilitic affections, and that "cortical epilepsy," as stated by Lang, of Vienna,* "is grounded more frequently in syphilis than in any other cause." Minute descriptions of such cases have been made by Charcot. Convulsive phenomena due to syphilitic poisoning are far more common among men than among women, occurring in the former usually between the thirtieth and fiftieth year, though there may be exceptions either way.

The writer has at present under his care a man of fifty years, whose attacks developed at forty-seven as the result of syphilis, the lesion being so extensive as to affect the frontal lobes in part, either organically or by implication, as shown by the occurrence of purely psychic attacks of the disease. He also has severe attacks of *grand mal*.

A young farmer of thirty years had frequent attacks of monoplegia and transitory motor aphasia as the result of the same cause. He most frequently suffered the loss of the use of the right hand and arm, and it was common for him to drop his knife and fork at the table, being able to use them again in a few moments and proceed with the meal as usual.

Almost immediately he was given vigorous antisyphilitic treatment, and these temporary monoplegias disappeared. His extreme mental sluggishness left him; he was able to go into the field and work, and he took an active interest in his surroundings. On stopping the treatment for two or three weeks, the spasms, the stupor, the inability to work, the drawling, hesitating speech, all came back, only to again disappear under another course of treatment.

I had never before, and have not since, seen a case in which mercury and the iodids so quickly and effec-

* "Twentieth Century Practice of Medicine," Vol. XVIII.

tually swept away, at least for the time being, the
results of syphilitic disease in the brain. When I last
saw the man at the end of a two years' course of
treatment, he was in excellent condition and on the
point of returning to his home cured. He had been
committed as insane because of marked psychic dis-
turbances due to the same thing that caused his
convulsions.

Bosworth * cites Weber as reporting two cases of
infantile syphilis in which epileptic convulsions fol-
lowed by coma developed coincidently with the cessa-
tion of the sanious discharge from the nose; the
nervous symptoms disappeared, however, immediately
upon the reestablishment of the escape of pus from
the nose.

Bosworth thinks it fair to assume septic infection
in such cases, though he appears to believe that the
convulsions are more likely due to the irritation of
the dried secretions retained in the nose.

Sir Morell Mackenzie refers to the same observa-
tions made by Weber.

Shock from Electricity.—In two cases that I have
seen, shock from electricity helped to establish the
disease. In one case the subject was a boy of fourteen
years, who had "some convulsions at the time of the
first dentition at seven months," and it might appear
that the electric shock during his fourteenth year only
served to again develop a disease that had previously
existed, though it is also fair to assume that the
attacks might never have recurred had it not been
for the electric shock.

The second case occurred in a man at the age of
thirty-two, who had long suffered from rheumatism,
having a marked degree of "rheumatoid deformans"
affecting the back. He never had a convulsion of any
kind prior to the electrical shock. He had measles at

* "Diseases of the Nose and Throat," 1893, p. 362.

five years and diphtheria at seventeen years; he was always temperate in his habits. Passing along the street, he saw a man, engaged in trimming an arc lamp, suddenly grasp the wries about the lamp firmly and stiffen out. The patient ran out to assist him and immediately upon touching the man through whom the current was going, he was thrown violently to the ground several feet away. He felt bruised and sore for several days, and shortly after had the first convulsion. His attacks are varied in character, being both *grand mal* and psychic. After the latter, which predominate, he is often automatic for half an hour or an hour at a time.

Heart Disease.—In a careful physical examination of 1070 cases of epilepsy due to all causes and embracing all ages, I was struck with the undue proportion of functional and organic heart lesions encountered, which were classified as follows:

Mitral regurgitation in	77
Irregular cardiac force and rhythm in	75
Cardiac hypertrophy in	50
Roughened first sound in	13
Systolic murmur at apex in	10
Aortic regurgitation in	9
Mitral stenosis in	2
Tricuspid regurgitation in	1
Diastolic apex murmur in	1

This makes a total of 238 who presented evidences of some form of heart disease or irregularity, though slight in many cases.

We must not, however, for a moment assume that the epilepsy in all these cases was due to the condition of the heart. On the contrary, in my opinion, we may lay the cause of many of the heart troubles to the effects of epilepsy, while in other instances they were present merely as coincidences.

I believe that epileptics are unusually prone to diseases of the heart and lungs; and whoever has the

opportunity for witnessing large numbers of epileptic
seizures of the worst type, especially during the tonic
stage, the stage of the single prolonged contraction,
when respiration is suspended and the heart is under
an enormous physical strain, will soon find himself
wondering why more epileptics do not die during the
seizure from the "*locking up*" of the respiratory act,
or from the forceful damming back of the blood upon
the heart, with consequent injury to that organ.

There are cases, however, in which the heart lesion
precedes and, in all probability, causes the epilepsy,
though such cases are rare.   Most of them occur late
in life, more frequently among men than women, and
they are nearly always associated with atheromatous
changes.   Some occur in childhood.

In all probability some of the cases of organic heart
trouble named above cause the epilepsy.

I recall two cases in which the subjects were
young, both boys, one nine and the other eleven
years, in which distinct mitral heart lesions existed at
the beginning of the epilepsy.   One had *grand mal*,
and the other *petit mal* attacks.   The father of one
was markedly alcoholic, the other rheumatic, though
neither patient, so far as could be learned, had ever
had rheumatism.

The first time I examined the one who had *petit
mal*, I had opportunity for witnessing a seizure.   I
had just asked the boy to stand in front of a window
that I might see his eyes better, and had taken hold
of both his wrists, when a sudden change occurred in
his expression, some drawing down and slight twitch-
ing of the muscles about the corners of the mouth, a
set, staring expression of the eyes, both pupils being
slightly contracted; there was a little hardening of
the muscles in both forearms, and the head was gently
turned to the right.   In less than two seconds the
attack had passed, and the boy was himself again at
once.

The pulse, which happened to be under my touch at the time, was not more than 50 a minute, while half an hour later it had gone up to its normal rate of 60.

The other boy had a similar heart, his pulse-rate during the inter-paroxysmal period being from 56 to 60.

The most rigid search failed to reveal any other cause for the disease in the two cases, both having good family histories, except as noted above, and we felt convinced that the heart lesion in each case was the cause of the epilepsy. This view was later strengthened after the condition of both patients had improved under treatment directed to the heart disease.

F. Savary Pearce * reports several cases which tend to reinforce the similarity between epileptoid phenomena and syncopal attacks that are independent of the epileptic neuroses, and calls attention to the full, slow pulse, with or without loss of consciousness, as tending to confirm the epileptic state.

Pearce is of the opinion that "organic disease of the heart rarely causes convulsions."

Chadbourne † deals with the subject *in extenso*, quoting Littré, Delasiauve, Tyson, Boyce, Gibbings, Bristowe, R. L. Jones, and others as supporting the belief that diseases of the heart may cause epilepsy; or, as some of these authorities state, particularly Balfour, pseudo-epileptiform attacks or spasms.

The English writers particularly tend to associate the slow pulse and epilepsy, while R. L. Jones reports a case "in which mitral regurgitation and slow pulse coexisted with epileptic attacks, and in which the infrequence of the pulse was thought to have been of neuropathic origin."

* "Annals of Gynecology and Pediatry," Nov., 1900.
† "American Journal of the Medical Sciences," March, 1902, p. 461.

Hebb* states that while he was listening to the heart of a patient twenty-nine years of age, who suffered from aortic insufficiency, he suddenly became aware that the heart had stopped beating, and immediately a general convulsion ensued which lasted several minutes. After consciousness returned, the patient talked rationally and coherently and arose without the slightest difficulty. He had suffered in like manner several times before.

George Lemoine in 1887 published an article entitled *L'épilepsie d'origine cardiaque et son traitement*, this writer being a most outspoken advocate of cardiac disease as a cause of epilepsy.

After carefully reviewing the work of many writers, Chadbourne reports eleven cases of cardiac disease associated with epilepsy, with autopsy findings in some of them, concluding as follows: "We have been unable in any of our cases to show to our satisfaction that there was any direct connection between the vascular lesions and epilepsy."

Notwithstanding the failure to establish a causal relationship between cardiac diseases and epileptic convulsions, the fact remains that we are still unable to say that such a connection does not exist. The true lesion, or the precise mechanism by which the fit is brought about in such cases may not be capable of material demonstration on the post-mortem examination. It may lie rather within the superior domain of some other field of science—perhaps in a combination of factors, like chemical pathology, like changes in the blood, or a functional neurosis confined to the heart alone, and irregularity in physiologic action—all founded primarily upon the diseased condition of the heart, or upon the same influence that wrought such disease, for we must not forget the possible influence of rheumatism or other constitutional states in this respect.

* "American Medicine," Vol. I, p. 174.

T. S. H., an intelligent business man of fifty-three years, of good physique, and fond of gratifying an unusually large appetite, remembers having a single convulsion at the age of eight years, which he says was caused by eating some kind of poisonous roots in the woods when he was looking for nuts.

He had no other convulsion until he was fifty-three, when, after a particularly hard day's work at the end of a busy season, one appeared, to be followed a year later by another similar in character, and six months later by a third, the latter occurring abroad, where the patient had gone for rest and recuperation.

I found his arteries as hard as whip-cords, those in the temples standing out in tortuous outline, barely compressible under the finger. There was a distinct murmur at the base of the heart, and he occasionally had symptoms of false angina.

His convulsions were attended with the utmost distress in the cardiac region, the patient declaring that he felt "every moment must be his last." This distress lasted in a lessening form from two to three days after the seizure.

The attacks were always *grand mal*, with the usual *grand mal* accompaniments, including the biting of the tongue. I could not rid my mind of the conviction that dietetic errors had much to do in the immediate excitation of the convulsions, though there was no question in my own mind as to the part played by the organo-neurotic lesion in the heart in producing the marked symptoms of distress followed by convulsions. The point in this case is this: Although this man had arteriosclerosis in a marked form, he would not apparently have had any epileptic convulsions had they not been immediately excited through some gastric disorder, for the correction of this condition vastly improved him in every respect, checking the seizures and lessening the cardiac distress.

Gowers states that an abnormal condition of the heart is met with in many cases, though not in sufficient number to indicate a definite relation to the disease, and that the imperfect supply of blood to the brain in diseases of the heart assists in the degradation of

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function and nutrition, but that the heart disease only becomes effective in producing epilepsy in the presence of congenital defect, or of a powerful, acquired disposition.

I do not doubt the force of the latter condition in the slightest; at the same time I believe that some forms of heart disease are capable of inducing epileptic phenomena, independent of predisposition of any kind.

The Influence of Sleep.—The idea of attributing to sleep the power of so often inducing epileptic convulsions belongs chiefly to Voisin and Lesague, the matter being treated thus by Voisin under *Causes déterminantes:* "Among the determining causes, sleep is without doubt the most frequent. All writers agree on this subject. Lesague was so much convinced that sleep was, so to speak, a component part of the epileptic attack that he expressed the opinion that any nocturnal convulsive attack was of epileptic origin. According to him, hysterical attacks occurred only by day."

To some extent what these writers say is true, though, in my opinion, they credit sleep with inducing the convulsion more often than facts seem to prove. In one series of 99,144 convulsions which occurred in 800 cases at the Craig Colony during the year 1902, the men had 29,754 during the day and 29,070 during the night; the women 24,649 during the day, and 15,671 during the night, making a total in the two sexes of 54,403 by day and 44,741 by night.

In some cases in which the attacks occur only by night, the influence is due to sleep itself, while in others the explanation is to be sought in the bodily condition—the state of the vital processes—at a period when physiologic resistance has reached its lowest ebb. In the latter cases, the attacks come on in the early morning between two and six, most commonly about three; in the former they appear as soon

as the patient falls asleep. We have seen such cases in which the sleep state always induced the convulsion, appearing even when the patient slept during the day; in other cases it appeared only at night.

A. Pick* gives some interesting measurements of the blood pressure during sleep, in an attempt to draw some conclusions from them with reference to nocturnal epilepsy. He makes the statement "that two-thirds of all epileptic attacks are apt to occur in sleep, most of them either with the first hour of sleeping or during the first hour before waking."

It seems logical to infer that nocturnal seizures must be due in large part to the silent processes going on even when we sleep, the processes concerned in the nutrition of the body.

There can probably be no fit without irritation of some kind in highly organized nervous tissue or centers somewhere, and seemingly the only avenues by which irritation can reach such tissue or centers during sleep are the blood and the sensory nervous system in some of its parts, particularly the parts of the latter that influence the action of certain viscera which are intimately concerned with the functions of digestion, absorption, assimilation, and reconstruction.

It is interesting to note a fact here, which we refer to more fully in the chapter on Treatment, namely, that while the number of attacks among the men by day and by night were almost equal—29,754 and 29,070, respectively—there was a marked disproportion in the same figures in the women, for out of a total of 40,320 seizures in this sex, 24,649 of them occurred during the day and 15,671 during the night.

SPORADIC CAUSES.

We may now briefly study a few factors that infrequently lead to the disease, among them being *hyper-*

* "Wiener medicinische Wochenschrift," 1899, No. 30.

trophy of the brain and *hydrocephalus*, which two conditions should not be confounded. The former may be general or partial. Its cause is obscure, depending, according to Virchow, on an increase in the neuroglia or connective tissue; while Rokitansky thinks the increased size is due to an increase in the immediate granular matter.

In two autopsies made by Andral, the white matter resembled "the white of an egg hardened by boiling." As a rule, such brains are anemic, and Andral states that the disease has two periods, the first in which the condition is chronic; the second, that of convulsions which generally end fatally.

Such brains on an average weigh from ten to fifteen ounces more than the average weight of normal brains.

Jacobi* speaks of this condition at some length, describing a case with convulsions which came under his observation.

Hydrocephalus was the cause in one case in the 1322 which have come under my care; the hydrocephalic condition was congenital, the convulsions developed at the eighth year.

This patient had a mild degree of internal strabismus, the treatment of which did not have any effect on his seizures.

Prenatal ossification, independent of such degenerative mental states as idiocy and imbecility, rarely causes epilepsy, though epilepsy is frequently found in connection with such mental states.

Jacobi, who has given much earnest study to the causes of epilepsy in the young, says: "Cases in which epilepsy of later years is due extensively to the compression of an originally normal brain in an abnormally compact and uniformly contracted skull, I have seen; but more are due to, or connected with, a premature partial synostosis. It is the fate of a great

* "American Medicine," Dec. 13, 1902.

many epileptics to have a comparatively small cranial circumference and an absolutely asymmetrical shape.''

Focal lesions of the central nervous system, independent of trauma, also play a small part in the production of epilepsy. These include lesions of vascular origin, such as softening, abscess, atrophy, sclerosis, and brain tumors, which in the majority of instances are either sarcomata or gliomata; the harder varieties that spring from bone and fibrous tissue are also sometimes found.

The form and degree of convulsions due to focal organic diseases in the brain depend upon the nature, location, and character of the lesion. This much may be said in general of epilepsies that follow such causes: They are apt to remain circumscribed in their field of action and rarely affect the mind to any appreciable extent, unless the *organs of the mind* are directly involved.

Asphyxia of the new-born has already been mentioned among the accidental causes of that period, but asphyxiation of other kinds may produce epilepsy, one man having come under my care who was poisoned by coal-gas and whose convulsions developed immediately thereafter.

Extreme fatigue may safely be put down as the determining cause of attacks in some who already have epilepsy, but we hesitate to claim that it alone ever produces the disease *de novo*.

I recall the case of a prominent merchant whose business kept him hard at work on Saturday nights until eleven o'clock or later. He lived across the street from my office, and five weeks in succession I was called between one and two o'clock in the morning to attend him in convulsions. On giving up the hard night-work, his attacks entirely ceased.

He was thirty-two years of age, and was a fine specimen of physical manhood; he had no harmful prac-

tices of any kind, but possessed a highly sensitive nervous organization and was subject to "nervous headaches."

Brietung* reports the case of a boy, sixteen years of age, who ran a long distance on a hot afternoon in July to take a cold water douche on his head, remaining under it a quarter of an hour. Soon after it was noted that he was unusually excited, and during the night which followed he showed great restlessness. The following morning an attack consisting of "jactitations, unconsciousness, etc," came on, which lasted some time, and was pronounced epileptic by the attending physician. From that time on the convulsions appeared from six to eight times a year, each being preceded by motor aura in the left hand and stimulation of the sexual impulse.

Brietung regarded the cold douche on the boy's head as a traumatism causing the epilepsy.

The train of symptoms induced by *phimosis*, congenital or acquired, may lead to reflex epilepsy, either through masturbation, of which we have previously spoken, or through excessive irritation.

I saw a girl nine years old whose epileptiform convulsions were due to a *stricture of the urethra*, as was proved by their cessation when the stricture was cut.

A boy had a convulsion at the age of one year which lasted four hours, and had no further trouble of the kind until he reached the age of eleven, when he was *vaccinated*, and his body became covered with a "general scaly eruption." The convulsions which appeared immediately after became permanent in character.

A young man, who had never had a convulsion before, was severely *stung by bees* about the face, and convulsions appeared with much regularity every two weeks thereafter.

* "Deutsche medicinische Wochenschrift," 1898, XXIV, 39.

It is well known that the sting of this insect is poisonous to some persons.

It is claimed that *hypertrophy of the Schneiderian membrane*, nasal and nasopharyngeal growths, renal and vesical calculi, and various forms of intestinal helminthes, from tinea to oxyuris, cause epileptiform convulsions. *Worms* were the ascribed cause in only two of the author's cases.

As to the influence of the latter in a few favorable cases in causing convulsions, there seems to be no doubt, though in such cases the cause probably lies as much in the general derangement of the intestinal tract as in the presence of worms themselves. It is unfortunate that *worms* are so often credited with causing convulsions. I have seen cases in which the treatment based on such a supposition did the child immense harm, while the spasms were found later on to be due to other causes.

I have never witnessed convulsions due to foreign growths in the nose, or to enlargement of any of its parts, though cases have been reported in which it was claimed that the removal of such agencies checked the convulsive attacks.

Such a case was reported by Grasskopf.* The patient, twenty years old, had suffered from convulsions for a year, which finally appeared several times a day. Treatment with the bromids did no good. There was suppuration in the antrum of Highmore, and some nasal polypi, and the patient had a seizure during the operation for the cure of these conditions, but none have appeared subsequently. The report does not state how long after the operation the patient became free from attacks, and it seems difficult to say which of the conditions present caused the convulsions.

* "Archiv. für Laringologie," XIII, 1; "Berliner klinische Wochenschrift," Aug. 4, 1902.

Richardson* reports the case of a boy with a hyper-sensitive nasal passage and marked hypertrophy of the middle and inferior turbinated bones, the relief of which checked the convulsions, which had not recurred eight months later.

Kahn† reports the case of a man subject to nose-bleed, who, on one occasion when bleeding was quite persistent from a small eroded spot, was locally treated by the application of a 10 per cent. solution of cocain, with the extraordinary result that the "patient became deathly pale and somewhat cyanosed; he had twitching of the facial muscles, and fell to the floor comatose; then he was seized with convulsions of the most marked epileptiform character, the con-vulsive movements affecting the right half of the body more than the left."

The author thinks that the anemic condition of the patient due to loss of blood may have contributed largely to the production of the fit, and states that convulsions from cocain have been reported both by Curgenven and by Garland, the former having wit-nessed convulsions after the ingestion of ten grains of the salt by the mouth; the latter, convulsions and death after fifteen grains, taken in the same way.

Eye-strain has long occupied debatable ground as a possible cause of epilepsy. In the large number of cases which it has been my privilege to study—some 1800 all told—I am unable to recall a case in which I felt the whole cause to lie in any anomalous con-dition of the visual apparatus.

Stevens's essay on " Functional Nervous Diseases," which was honored with a prize by the Royal Academy of Medicine of Belgium in 1883, contains recitals of ocular conditions found in 140 cases of epilepsy, which he described as follows:

* "Medical Record," July 14, 1900.
† "Medical Record," March 24, 1900.

"The general results of these examinations have been to reveal the existence of refractive anomalies in a considerably greater proportion than has been found by Cohn in his examinations of the eyes of school children, or by other observers in similar investigations in Germany, Russia, and America."

In 100 consecutive cases, Stevens found hypermetropia, with hypermetropic astigmatism in 59; myopia with myopic astigmatism in 23; and emmetropia, or refractive errors less than one dioptric, in 18.

Stevens claims, in the same essay, that muscular insufficiencies of the ocular apparatus are potent factors in the development of epilepsy, bringing forward a number of instances in support of this view.

Ranney* refers to work done by himself in 1897 in preparing a special volume on "Eye-strain in Health and Disease," in which he makes some very remarkable statements concerning the good results secured in treating epilepsy through the eye alone.

Gould† is likewise convinced that eye-strain has an important place in the etiology of epilepsy, and in the chapter on Treatment we refer in detail to the work of this distinguished ophthalmologist at Sonyea in the treatment of sixty-eight cases through the careful correction of errors in refraction.

In "American Medicine" (July 5, 1902) Gould refers to six cases of epilepsy due to ametropic eye-strain, in which correction of the condition resulted in apparent cures in some, in improvement to others.

He says: "That epilepsy has been caused by imbalance of the ocular muscles and cured by operation has been asserted, but this cannot be true, if, as we believe, the incoördination of the external ocular muscles is, in itself, the result of ametropia. That

* "New York Medical Journal," Nov. 22, 1902.
† "American Medicine," Vol. LV, No. 1, p. 2, July 5, 1902.

ametropia does occasionally cause epilepsy is, I think, beyond question by those who have studied the facts with a genuine scientific spirit.''

There should be some significance in the fact that not once do we find *eye-strain* or, indeed, visual defect of any kind, in the histories of 1324 cases admitted to the Craig Colony in eight years, either under '' assigned cause '' or under ''probable cause.''   It may be claimed by some that general practitioners and neurologists are not good oculists and might not be able to locate the cause of epilepsy in the eye, even if it were there.

To some extent this might be true, but the tendency of the day is so strongly toward specialization, and epilepsy is so difficult to treat in a way to yield satisfactory results, that a large number of such cases, prior to commitment to an institution such as the Craig Colony, are subjected to the crucial tests of the ophthalmic surgeon as well as to specialists along other lines, in the efforts made to determine the cause of their attacks.

CHAPTER VI.

SEIZURE TYPES.

The Relative Frequency of Different Seizure Types. Descriptions of Regular and Irregular Forms of Each. Grand Mal. Petit Mal. Psychic. Jacksonian. Serial Attacks. Reflex Epilepsy. Epileptic Equivalents. Partial Epilepsy. Tetanoid Epilepsy. Hystero-epilepsy. Myoclonus Epilepsy.

WHEN considering the nomenclature of seizure types based on symptomatology, reference was made to some of the more prominent features of the many forms of epileptic convulsions, though not to an extent sufficient for diagnostic purposes.

Before we take up the important matter of diagnosis, we should possess clear ideas, not only of the more prominent and regular forms of the disease, but of the less prominent and irregular forms as well.

I know of no studies made to determine the comparative frequency of seizures of different types, and while it cannot be claimed that the results obtained in 1325 cases studied in this respect are conclusive, they are offered as quite accurately indicating the comparative frequency of each.

Seizures of the *grand mal* type are far more common than any other. In the 1325 cases mentioned, 815 of whom were men and 510 women, 482 of the former and 292 of the latter suffered from this form, making about 60 per cent. of the whole number who had *grand mal* convulsions, the proportion in the two sexes being substantially the same.

In the same cases, 38 men and 34 women, constituting about 4½ per cent. of the former and 6½ per cent. of the latter, had *petit mal* alone; therefore it appears

that more women than men suffer from this lighter form of the disease.

Under the purely psychic form, the total number is still less, as four cases only, three men and one woman, are classed under this head. This does not, however, by any means indicate that this type of epilepsy is as rare as this would seem to make it, for among the *mixed* types to be noted later on, it is comparatively common; that is, the same individual has *grand mal* and psychic, or *petit mal* and psychic, as the case may be, the former combination being considerably more frequent.

The four cases of purely psychic epilepsy mentioned were uncomplicated by attacks of any other kind. We should also bear in mind that the unobtrusive character of psychic epilepsy makes its presence possible for an indefinite period of time before its existence is known to any but the patient himself, and doubtless many cases of it go continuously unrecognized.

Jacksonian epilepsy is nearly twice as commonly met with as psychic epilepsy; there were nine such cases, six men and three women, among the 1325 studied.

It is my belief that Jacksonian epilepsy, in which the attacks not only begin, but end as such, is extremely rare. It is not, however, an uncommon thing to find attacks that assume this form in the beginning only to run into general epilepsy before the fit is over. In the nine cases mentioned here, the type was preserved pure throughout the seizure.

Cases in which the convulsions are mixed in character stand next in frequency to *grand mal*, and we note that 286 of the 815 men and 180 of the 510 women —constituting in the two sexes combined about 32 per cent. of the entire number—suffered from mixed forms of the disease.

There is no fixed rule or order observed in the mixing

of types in the same individual, though, as we have previously stated, *grand mal* and *petit mal* are more often noted together than any other, the very probable reason being the greater frequency of these forms individually over the other two.

It must also be borne in mind that *fixity of type* is not constant. An individual may suffer from *grand mal* attacks for a long period and then have some other form, though changes in this respect are more apt to be from the lighter to the more severe, as, for instance, from *petit mal* to *grand mal*, or from Jacksonian to *grand mal*. I do not recall a case in which Jacksonian seizures were replaced by those of a psychic nature; nor have I ever known the reverse to be true. At the same time, such a possibility cannot be denied.

ILLUSTRATIVE CASES OF GRAND MAL EPILEPSY.

Under this I shall describe both regular and irregular forms of the disease, assuming that the regular or classical convulsion means the invariable presence of certain features during the attack; the irregular, the absence of or a marked variation in any of the pathognomonic symptoms present under the former.

Case I.—M. J. O'T. was sitting on a chair when the attack began, the first thing unusual being a short, low, moaning sound, like a sigh. The patient then moved forward to the edge of the chair as though about to rise, and remained "fixed" in this position for thirty seconds; during this time his face was pale, his eyes were set and staring directly forward, and both hands opened and closed in rapid alternation. Then his eyes began to move slowly upward and outward to the left, and when they had completely turned, his head began to turn in the same direction and went around until the chin was almost directly over the left shoulder. The convulsive movements in the hands now ceased, and both remained tightly clinched in clonic contraction, the thumbs being tightly flexed

across the palms with the fingers over it.   Suddenly
the body lurched forward and fell to the floor, striking
partly on the left side which was due to the force of
the contractions in that direction.   Immediately on
striking the floor, both arms were drawn upward and
apart, the forearms being flexed on the arm, and the
contraction being more marked on the left side than
on the right.   Both legs were drawn upward and wide
apart at the knees, and the lower legs were sharply
flexed on the thighs, this condition being more marked
on the left than on the right side; both feet were
turned inward at the ankle, this also being more
marked on the left than on the right side.   The
mouth was open, and the lower jaw was drawn forc-
ibly downward and strongly to the left.

The patient remained immovable in this position
during the stage of tonic contraction, which lasted
seventy seconds, when the stage of clonic contraction
set in, which was very brief, lasting twelve seconds;
there were only three shock-like contractions during
that time, these appearing most marked in the ex-
tremities, and of equal violence in all parts; all con-
vulsive movements ended with the last of these.

The period of stertor that followed was prolonged
and severe, and covered three minutes, during which
time a large amount of froth was blown from the mouth
at each expiration, the cheeks being forcibly drawn
in and forced out with each inspiration and expiration.
When these movements ceased, the patient passed
into a comatose sleep that lasted fifteen minutes,
after which he sat up and was able to talk in a confused
way.   While there was no mental disturbance beyond
this slight temporary confusion, he was greatly ex-
hausted physically and felt indisposed for any activity
for several hours.

In this case the tongue was not bitten, there was
no aura of any kind, nor was the so-called epileptic
cry present; the only sound which escaped the patient
was the low moan at the beginning—a significant
involuntary expression of distress.

Case II.—J. B. was sitting on a chair when his face
was observed to grow suddenly pale, especially about
the mouth.   The blanching grew more pronounced

during a period of two minutes, and the patient remained motionless and silent during that time.

Suddenly he threw the right arm above his head and, without any cry, fell to the floor. The convulsive movements began in the right hand, the fingers closing on the palm in severe clonic contractions, the thumb being inserted at each contraction between the index and second fingers. Then the convulsive movements appeared in the forearm, next in the arm; then they involved the head, passed from the head to the left hand, then up to the left forearm and arm, jumped next to the left foot and passed up the leg to the body. The left hand contracted in a natural way, the thumb being outside the fingers just as the fist is closed preparatory to striking a blow. The head was turned to the left, and the mouth was drawn in the same direction, the jaws opening and shutting with great violence.

The eyes were turned upward and to the left, when suddenly a single, sharp convulsive movement turned the head to the right, the mouth and eyes being drawn in the same direction; now convulsive movements began in the foot, and rapidly passed up the leg, but being at no time as severe as in the other parts previously affected.

The period of tonic contraction lasted thirty seconds, while the clonic contractions, which were general, consisted of a series of shock-like movements, affected the entire body at once, and lasted forty seconds; the right arm was disturbed by disappearing contractions for a full minute after muscular composure was elsewhere complete.

The face was deeply cyanotic to the end; the period of stertor was prolonged; the pupils were widely dilated throughout the seizure and unresponsive to light. The salivary flow was light; the tongue was not bitten; the urine was not voided; the temperature was 98.8°; the pulse 68; and respiration 22; all this was noted immediately after the convulsive stage was over, and before the stage of coma.

The patient aroused from the comatose state in four minutes, then slept soundly for twenty minutes, after which he was substantially in his normal state.

Case III.—C. S. was engaged in making a bed when she suddenly stopped, straightened herself up and ran five yards, when she fell to the floor. She was able to speak at the time, and complained of a "crawling sensation" in the pit of the stomach. The sensation appeared to extend gradually upward until it reached the throat, when a feeling of constriction was experienced, and the patient made a single, loud, sharp cry, and closed tightly both eyelids at the same time.

Her face wore a terrified aspect during the moment preceding the involuntary closing of the eyes. The convulsive movements then appeared, beginning in both eyelids and extending to the eyeballs, which became involved and turned sharply upward and to the left. The face began to assume a deep cyanotic appearance. The left hand was clinched with the thumb over the fingers, the right partly so, the thumb being caught between the first and second fingers.

The legs were flexed on the thighs, with the knees wide apart. This position of tonic contraction lasted for eighteen seconds and was followed by twelve sharp, decisive, clonic spasms, during which the tongue was protruded between the teeth and severely lacerated. At the end of the clonic spasms the patient sank into deep coma which ended in sleep that lasted for three hours. On awaking she was clear mentally, being able to recall all that transpired at the beginning of the attack up to the time the choking sensation reached the throat, just prior to the cry; after which all was a blank until she awoke from the sleep three hours later.

As soon as the active part of the attack was over, the temperature was 99.5° F.; the pulse 103, and respiration 22. She complained of great thirst, general muscular soreness, headache, and nausea. The thirst was due in part to the profuse sweating, this phenomenon being at times excessive just after the periods of contraction and stertor, in part to the mild degree of post-convulsive fever, which, as a rule, is of short duration, rarely lasting over two or three hours, and generally subsiding in less time than that.

Case IV.—E. Mc. was sitting on a chair with her arms folded, when she suddenly screamed, threw both arms

over her head, and fell face downward to the floor. There was no aura of any kind, the first thing noticed being the cry, and the patient simultaneously falling to the floor. She was immediately turned on her back, and it was noted that the face was pale and both eyes were tightly closed. Almost at once they began to open, turning upward and to the left at the same time; then convulsions began in the left leg, continued up that side, and involved the left hand and arm; they next appeared on the right side of the face, drawing the mouth open and upward to the left side. The face became more deeply cyanosed, the pupils of both eyes were widely dilated, and the corneal reflex was entirely lost. Both legs were sharply drawn up; the legs flexed on the thighs, and the thighs on the abdomen, the knees being wide apart. The left arm was flexed tightly against the chest; the right forearm flexed only part way on the upper arm; both hands were tightly closed, the thumbs lying over the fingers.

In this position the tonic spasm lasted seventeen seconds, while the clonic period that followed lasted twelve seconds; the convulsive movements of this period shaded out to complete composure and terminated in a few deep respiratory movements which expelled froth from the mouth. At the end of the stertor period, the temperature was 99.2° F.; the pulse 122, and respiration 26. Urine was involuntarily voided and the patient remained in a comatose sleep for thirty minutes; then she sat up when spoken to, but was stupid and indifferent to her surroundings. The tongue was slightly injured during the clonic contractions.

Case V.—F. C. was standing unoccupied, when he suddenly staggered backward two or three steps, falling heavily to the floor on his back. The first noticeable contraction occurred in the right thumb, extended next to the right hand, then to the right leg; after which it passed over to the left side involving the hand first, then the arm, then the leg, and finally the face, when a typical epileptic cry was given.

Both fists were tightly clinched, and both thumbs were inserted between the first and second fingers. This has been called the *epileptic hand*. Both eyes

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were rolled up and to the right; both pupils were widely dilated, and the conjunctivæ were congested.

During the clonic stage the lids opened and closed spasmodically. The face was deeply flushed, the color shading into a darker cyanotic hue as the tonic period became prolonged.

At first the arms were extended almost at right angles to the body, then drawn in and flexed tightly across the chest. The legs were flexed on the body. The tonic part of the convulsions lasted forty seconds, followed by the clonic, in which the movements of the limbs were excessive, the patient being violently tossed about through the force of the shock-like muscular contractions. This period lasted twenty-five seconds, respiration being re-established by a series of deep, stertorous, blowing sounds. The tongue was severely bitten during the clonic spasms, and the froth which was expelled with each noisy expiration was tinged with blood.

Immediately after the stertor period the temperature was 99° F.; the pulse 135, and respiration 28. The patient fell into a deep sleep which lasted two hours and a half, and on awakening complained of a severe headache and showed great irritability of temper.

These cases suffice to give a fair idea of the *grand mal* attack. We note from them that the convulsive movement may begin in almost any part of the body, though it most often begins in the hand or leg; that the eyes are always involved; that the face is cyanotic, this being so extreme in some cases as to cause it to be almost black. All reflexes are abolished. There are three distinct periods: That of the tonic contractions; that of clonic contractions; and that of stertor and coma. All of these are necessary accompaniments of every *grand mal* attack.

The direction in which the patient falls indicates in a measure the location and severity of the muscular contractions. Some always fall straight forward; others to one side or the other; others backward; while still others sink gradually down just where they

stand, though these are rarely true cases of uncompli-
cated *grand mal* attacks.   Some fall so persistently
and repeatedly in a manner to strike the same part
of the body every time, like the occiput, the outer
angle of the eyebrow, or the point of the chin, that
it is a part of the treatment to protect these places
with pads to break the force of the fall.

The epileptic cry is not essential in every convulsion,
and when it appears it is due to one of two things,
very possibly to both: To some convulsive disturbance
in the speech center, or to the forcible expulsion of air
from the chest, through the contraction of its muscular
walls, the rushing of the air through the vocal cords
producing the inarticulate sound.   Injury to the
tongue is an unessential incident.   When it is present,
it means that the tongue was protruded between the
teeth during the period of clonic contractions.   When
not present, it means the absence of muscular action
that causes protrusion of the tongue while the jaws
are opening and closing spasmodically.

ILLUSTRATIVE CASES OF PETIT MAL CONVULSIONS.

We come now to the consideration of seizure types
that are distinguishable from the form just described
only through their lessened severity.   They partake
of all the features of *grand mal* epilepsy, including
the loss, or disturbance, of consciousness, and general
muscular commotion.   But though they are less
severe, they have the appearance of disturbing the
mental faculties more; and this is true, for while it is
disturbance only in such cases during certain stages of
the convulsive period, there is total abolition of all the
mental faculties in the severer forms at the same stage.

In *petit mal* attacks we may be prepared to
note: 1. The mild degree of muscular disturbance or
commotion, as compared with that present in *grand
mal* attacks.   2. The slower invasion and lesser degree

of involvement of the faculties of the mind and the quicker return of the patient to his normal state after the fit is over.

Case I.—R. E., a robust, well-developed young man of twenty-five years, was standing idle when the attack began, the first thing noticed being a marked pallor that covered the entire face. Presently a number of large, irregular, dark blotches appeared, most noticeable about the sides of the face and on the neck. These blotches soon became of a dark, livid color, but were not raised above the surface of the skin.

The patient stood motionless for a minute, the pallor and the blotches all the while growing in prominence. Then he suddenly called to the nurse who stood near, and before the nurse could reach him, cried out, "Oh! Oh!" and started to fall somewhat to the left. The first muscular movements noted were in the eyes, both of which rolled upward and the eyelids twitched convulsively. Next the mouth was drawn to the left in clonic movements, repeated six times. The movements next appeared in the right hand and arm, then the left hand and arm, then the left side of the face, after which both lower extremities were involved in several spasmodic jerks. There was no tonic period, all the contractions being clonic, except the first in the left side of the face; the twitching of both eyelids continued throughout. Both pupils were dilated during the entire time, and were unresponsive to light. Neither hand was firmly contracted, the fingers of both being slightly flexed.

The progress of invasion was slow and deliberate throughout. There was no flow of saliva; no injury to the tongue. The comatose period lasted two minutes, when the patient arose, looked about in a dazed way, and appeared to be apprehensive of danger, as was indicated by his quick, nervous movements and his apparent desire to leave the spot. This disturbed period lasted ten minutes, when the patient suddenly grew calm and docile and was able to answer questions in an intelligent manner, though not with his usual readiness. He said that he felt an attack coming by a "queer feeling" in his stomach, and that he did not

lie down because he had often felt that way without having an attack.

Temperature, pulse, and respiration were normal after the attack, and there was no involuntary action of any bodily functions.

Case II.—M. V. was sitting down when it was noted that his pupils were beginning to dilate. Immediately his head began to turn slowly and almost imperceptibly to the left. While this was going on, the patient got up, walked once about the room in a purposeless way, opened a door, stepped out on the veranda, and closed the door after him; almost immediately he re-opened it and entered the room, and took up a standing position near the nurse employed in his care.

In the meanwhile, the patient's head had become turned completely to the left, and slight convulsive twitchings were observed in his face, being most marked on the left side. Shortly after these had set in, saliva began to flow from the mouth, though it was not frothy and there was no blood in it. All this time the patient had been fully unconscious, having been so since the beginning of the attack, all his first movements about the room and out to the veranda, and his return having been made without his knowledge. With the fading of the convulsive twitchings in the face, the head started to regain its normal position, and the first indication of returning consciousness was when the patient sought the toilet room and voided his urine. On being asked immediately what the trouble was, he replied, "I guess I have just had a little spell."

The seizure lasted three minutes in all, and the bulk of it was made up of disturbed or lost consciousness; there were only the slightest convulsive movements, confined to the face, while the tonic period was entirely absent.

The localized spasm in the face in this case and the absence of any cry, or fall, might have tended to confuse it with Jacksonian epilepsy, but the single fact that consciousness was completely lost in the very beginning was sufficient to distinguish between the two, for in true Jacksonian fits consciousness is

often but little disturbed—sometimes not at all; while convulsive movements always precede any disturbance in this direction.

Many Jacksonian epileptics are fully capable of witnessing their own seizures, getting clear enough impressions to remember them, so that they can fully describe them in detail after the attack is over.

In this case there was no elevation of temperature, or any increase in the pulse or respiration after the seizure was over.

Case III.—C. K. complained of feeling "generally miserable," and showed great irritability of temper; he wandered about the house in an aimless way, entirely at variance with his usual custom.

Finally, while walking across the floor of the smoking-room, the attack came on. The patient staggered backward about ten feet, stumbled, then sat on the floor as if some one had forcibly thrown him down in that position. He began to swing both arms about his head, then lowered his hands to the floor, using them to shift himself about, as though he were deprived of the use of his legs and feet and depended on his hands for locomotion. Next, his face became deeply congested, then cyanotic; this quickly passed and changed into intense paleness. Both pupils were widely dilated. The left side of the mouth and the muscles about the left eye were mildly convulsed, the face being drawn somewhat to the left. He remained in the sitting posture and was unconscious ten minutes.

Presently he arose to his feet, and his face assumed a look of the most abject terror; he began walking about the room, mumbling incoherently, and apparently trying to make the nurse understand something he was incapable of explaining in a lucid manner. This confused condition lasted for half an hour before his normal state was re-established.

Case IV.—A. L. S. was standing quietly when, without warning of any nature whatever, she suddenly fell on both knees; her face grew pale at once, and both eyes were set and "apparently stared at some object on the floor immediately in front." Evidences of slight muscular twitchings were noted about the throat; the last of these terminated the seizure, and

the patient was able to get up immediately after that. The entire attack did not last more than fifteen seconds.

On regaining her feet she walked about for three minutes in a confused, automatic way, when she passed into her normal condition, and complained of a feeling of great exhaustion. On being questioned as to what she remembered just prior to the onset of the attack, she declared the last thing she recollected was "a vision of a score of repulsive faces all coming straight toward her." In this case none of the bodily functions passed from under control. There was no aura of any kind; no cry, and no biting of the tongue; while the temperature, pulse, and respiration remained normal.

Cases in which patients are attacked in this way, *i. e.*, when they suddenly sink to the floor through the simultaneous loss of power in both legs, are rare, and indicate a lesion in the internal capsule of a nature to impair all the fibers of the motor tract. The problem in such cases is the mechanism by which consciousness is so quickly and so sharply lost.

Case V.—C. S. was engaged in making a bed when she suddenly collapsed, falling quietly in a "crushing down" way to the floor. (This manner of falling is typical of this form of epilepsy and differs wholly from the fall of the *grand mal* seizure. It is more like a general giving way, a gradual collapse, than the sharp, accentuated fall that occurs when all the muscles are suddenly caught in a tight, vise-like grip; the shock of the contractions being great enough to throw the individual with extreme violence to the ground.) As soon as she touched the floor both eyes were observed to roll upward; the lids were wide open, and the expression was set and staring; the pupils were widely dilated. The first muscular twitchings occurred about the eyes, being equally well marked on both sides. Next the left arm became slightly convulsed, and at the same time drawn up across the chest; the fingers and thumbs flexed were in the scoop position. The knees and elbows were flexed; the legs were drawn up part way, and the left arm was more bent than the right. The patient remained in this position fifteen

seconds, when she raised herself to a semi-recumbent one and began feeling about the floor as though searching for something, this action being purely automatic.

Then began a distinct smacking of the lips, the so-called *tasting movements* that are, not infrequently, an accompaniment of the active part of the epileptic state. On the subsidence of this, she began to talk incoherently, but was almost immediately conscious of her surroundings. The entire attack had lasted three minutes, and it was characterized by the absence of any cry, any injury to the tongue, any distinct convulsive movements, tonic or clonic; any loss of control of bodily functions, and, finally, it was not followed by any period of stertor or comatose sleep. There was in it simply a period of muscular incapacity, combined with a sharp loss of mental ability; the former lasting less than a minute all told, the latter for fully three minutes, thus confirming the statement previously made, that mental disturbance is more often a characteristic of this light form of epilepsy, while mental abolition is more often the concomitant of the severer forms of the disease.

ILLUSTRATIVE CASES OF PSYCHIC EPILEPSY.

The clinical manifestations of this form of epilepsy are not nearly so complicated as those of the forms just discussed, for the reason that they have to do with the mental activities of the individual only; never in any case passing over and involving the motor side in any manner or degree. It is this fact which gives psychic epilepsy its interest, and, in the absence of any knowledge of the mechanism by which the conscious life of the individual is suddenly snuffed out, without even the appreciable tremor of a muscle infinitesi-mal in size, and of the manner in which it is as suddenly and as completely restored, we can at this time only marvel as at something we do not understand.

Epileptics themselves refer to such attacks as "weaknesses," "dizzy spells," "blanks," "faints,"

"absences," "darknesses," etc., applying these terms in some instances to the lighter attacks of *petit mal.*

Some who suffer from psychic and *grand mal* seizures at the same time, do not regard the former as epilepsy at all, feeling that all the concomitants of the *grand mal* fit must be present before there can be a real attack.

Irrespective of this, psychic epilepsy is a distinct form of the disease which at present is but little understood, and which, because of the unobtrusiveness of its character, often exists for years before it is recognized.

The cases reported here came under the writer's personal observation and are typical illustrations of their kind.

Case I.—C. S., a man of forty-nine years, had been an epileptic of the syphilitic type since the age of forty-seven, his attacks as a rule having been general and of great severity, though he was accustomed to occasional attacks of a psychic nature also. At one time he had been employed, in an adjoining room, in puttng pamphlets into envelopes and addressing them from a printed list, afterward going through bundles of them done up for the mail to see that they were all correct. I speak of this to show how this habit of precision, was carried over into the automatic psychic state that followed.

The patient was engaged in tying the pamphlets into bundles when he suddenly started up and came hastily into the room I was in. The expression of his face, which was white and tense, indicated that he was in a seizure. Both pupils were dilated, the eyes having a set, staring expression, and the appearance of looking with a strained gaze into space without seeing anything.

I was going over some papers in the safe at the time. He came and quietly looked over my shoulder for possibly ten seconds, then turned and entered a closet, where he picked up some pamphlets, counted them, adjusted them in an orderly bundle, and laid them

back on the shelf. He had been getting pamphlets from this closet for several days, counting and arranging them each time he took them out, just as he did on this particular occasion.

I took him by the sleeve and led him to the window. He came without resistance, but with a puzzled look on his face. His pupils were insensible to light or shadow, and the corneal reflex was much blunted, as was sensation all over the body. He recoiled a little when stuck with a pin, provided he saw the act; otherwise he hardly seemed to fell deep penetration. I showed him my watch and asked him the time, having to shout quite loudly to make him understand. He promptly answered, "Twenty-five minutes after nine," which was correct.

I motioned to him to take a seat, which he did at my desk, when he adjusted his glasses and began to go over some papers in a businesslike way, taking up one batch of them after another, examining each carefully, and putting them all back exactly as he had found them. He then got up, and in walking about the room, saw the telephone wire where it came through the floor, took hold of it, and gave it a vigorous pull. I told him not to do that, shaking my head at the same time. He said, "Oh! it must come up; it must come up"; but he dropped it without making another effort.

The condition of automatism lasted fifteen minutes, and pulling at the wire was the only foolish thing he did. In all other respects he was calm, obedient, and orderly in every way, and it might have been hard for the uninitiated observer to believe that the patient was having a psychic seizure.

Suddenly he returned to consciousness, glanced quickly about the room, seemed confused at finding himself there, and at once returned to his work. Half an hour later he declared he had never before seen my watch; that he had absolutely no recollection of being in my office; though he knew he had passed through an attack on account of the headache that followed.

A week later he had a very violent *grand mal* attack, in which he fell to the ground and fractured his nose.

Case II.—A. L., a woman, frequently had "weak-

nesses." When engaged in conversation, she would suddenly hesitate as though trying to find the word she wanted, and a slight pallor would sweep over her face at the same time. She would then stop talking, and in ten or twelve seconds would take up the conversation just where it had been left off. Usually no ill effects followed, not even a headache.

Case III.—E. S., a woman of unusual intelligence, was sitting at a table writing a list of names. I happened to be watching her at the time and noted that she suddenly changed color, but kept on with her work. After she had written the same name and address six times, she regained consciousness and appeared much embarrassed at what she had done, knowing that she had repeated the names while under the influence of a seizure.

Case IV.—P. DeM. while paring vegetables would suddenly let the knife drop from his hand and sit motionless for from five to ten seconds, then pick up the knife and go on with his work. At other times, while playing cards, for instance, he would suddenly assume a blank expression, take out his watch, study it carefully, call out the correct time and say he must be about his work. Suiting his actions to the word he would go to a closet on the floor above, and get his broom and dust cloth and begin to work at the cleaning he is accustomed to do regularly each morning, always looking at his watch before starting it.

On one occasion he began cleaning at 9 o'clock at night, keeping at it half an hour before consciousness was restored. On this occasion he said just as the attack came on, "It's 7.30 and I must get to work." The time actually was 9 P. M., 7.30 being the hour he was accustomed to do the work each day.

When fully in the attack, the nurse spoke to him, shouting quite loudly in his ear to make him understand, asking him what time it was, and the reply he gave was correct; he had absolutely no recollection afterward of having been asked the time or of having given it.

In the "Journal of Nervous and Mental Diseases," August, 1902, the author reported the case of a com-

mercial traveler who was subject to periods of automatism, one such period having lasted twenty-eight days. During this time the man traveled quite extensively in the West; visited his customers; wrote orders; sent telegrams; and engaged in various business transactions that he afterward had no recollection of whatever. The only way he could be convinced that he had done all these things was through reference to copies of all letters and orders he had written, and through daily entries in his diary, in which he had noted his condition with exactness and regularity, using a certain Greek sign to designate his epileptic attacks, as he did not want others to know that he was a victim of the disease.

Near the end of the automatic period, he had two or three *grand mal* seizures and utterly collapsed, necessitating his restraint in a hospital.

In addition to these purely psychic cases, which involve no motor element whatever, which do not even disturb the fine adjustment and regularity of the muscles used in writing, there are other cases, perhaps a little more numerous, in which motility is abolished during a very small space of time. These do not display any convulsive movements whatever, the muscles simply becoming momentarily limp and inactive, regaining their power of contractility coincident with the reestablishment of consciousness, as the following cases show:

L. DeR., while combing her hair, suddenly let the comb drop to the floor, both arms falling quietly to her sides in a natural position. She stood immovable in this position for a few seconds, stooped and picked up the comb and went on with its use as before. She made no outcry or movement of any kind; there was no twitching about the face; no turning of the eyes; no biting the tongue—nothing but the sudden relaxation in both hands and the mental blank, both of

which disappeared as suddenly and as completely as they came, leaving nothing to indicate that a seizure had occurred.

M. P., while sitting at the table, suddenly dropped her knife and fork, letting both hands fall in her lap. She leaned back in her chair, with her eyes closed, and the eyelids tremulous and the face pale; she remained in that position not exceeding seven or eight seconds, and suddenly regained control of herself and began the meal again. In this case the disturbance of normal muscular tonicity was a little more general than in the case last described, though there was an entire absence of muscular contractions of any kind or degree.

In addition to these clinically distinctive cases of psychic epilepsy in which motility is fully preserved, and others in which motility is locally lost during the unconscious period, there is another grade or class of psychic attacks that might properly be termed the *epileptic dreamy state*. In this state there is not, so far as it is possible to judge, any alteration in motility, but an alteration in consciousness only, the faculties of the mind not being lost, only disjointed, dissociated, and beyond the individual's control.

L. Pierce Clark reports a case of this kind in the "Medical Record" for February 20, 1897; the case being that of a woman who had a feeling of constriction across the forehead just before the beginning of the attacks, during which her conception of the arrangements of the room in which she happened to be, and of the people who chanced to be near her at the time, is different from that which she has while in her normal state. She is able to converse at such times, but declares that she is not entirely conscious of what she says, and is apt to express ideas entirely contrary to those she really entertains. When this condition has passed, she remembers what she said while in this

partially subconscious state and endeavors to correct wrong impressions made at that time. She also suffers from *grand mal* and *petit mal* attacks, though the attacks described above are far more frequent in occurrence than the rest.

Clark reports another case in which the patient may be in an attenuated psychic state when spoken to, and will fail to answer, but when the attack is over she is certain to ask you to repeat your question. She declares that her attacks "come and go like waves." Still another patient has attacks so light and transitory that he fails to read part of a line or part of a sentence; suddenly realizing that he has lost the sense of the subject, going back to find the missing link which is all new to him.

In every way these slight degenerate phases of disturbed consciousness are of the greatest interest, for the line that divides them from the normal is at times perilously indistinct, and I have no doubt but that they may and do exist for years, even sometimes through life, without detection; and it is interesting to speculate as to whether the process involved in the production of these states may not be identical with the process underlying the minute, forced memory accessions that many, perhaps most, people experience some time in their life. By accessions to memory I mean the sudden interjection into the field of consciousness of some insignificant act or thing experienced by the individual years ago, the memory of which had never been revived, its revival eventually being what might be termed a psychologic accident.

ILLUSTRATIVE CASES OF JACKSONIAN EPILEPSY.*

In this form of epilepsy we have again to deal with disorders of motility, localized always, though not necessarily remaining so throughout the seizure, the

* See "Partial Epilepsy" for more complete description of Jacksonian epilepsy.

feature of motility differentiating the fit on a very distinct basis from the psychic epilepsy just described.

Case I.—A. J. was sitting on a chair when she complained of a "tingling" sensation in the left arm, which she grasped quickly with her right hand. The fingers of the affected hand were flexed on the palm, the thumb being underneath, and the hand flexed on the forearm.

While the patient was grasping her left arm, she was spoken to by the nurse; she looked up and smiled, though made no reply. After the convulsion had passed, as it did after three or four simple shock-like jerks confined entirely to the arm, the patient was able to recall and describe all that transpired while her arm was in contraction.

She grasped the arm with the idea of checking the extension of the seizure. There seems to be ground for believing that this is possible in some instances. Tying a string or a handkerchief tightly about the upper arm when the attack begins in the fingers sometimes has the same effect.

Case II.—C. W., a man of fifty years, a subject of brain syphilis, frequently has attacks that are confined wholly to the muscles of one side of his face. During these, if the contractions are not too powerful, he can talk well enough to be fairly well understood. At other times the seizure spreads and involves one arm. In either case, after the fit is over the man is able to describe all that transpired about him while the fit was in progress.

Case III.—J. P., a boy of eighteen years, was subject to both *grand mal* and Jacksonian epilepsy; individual attack which began as the latter, extending at times into the former; while at other times the Jacksonian type was preserved pure throughout.

As he was starting to go from one building to another, it was noticed that he stopped and remained motionless for some seconds, when the right arm began to be violently convulsed, the fingers being sharply flexed on the palm, the thumb held down by them. The nurse approached him, thinking he might fall, but he showed no indication of doing so. On

being asked what the trouble was, he replied, "I—
am—having—a—fit." He was then told to sit down,
but answered, "No,—I'll—be—all—right—in—a—few
—minutes."

The disturbance passed completely in a few seconds
and he was himself fully at once; just, indeed, as he
had been throughout the seizure, consciousness being
at no time impaired, much less lost. The contractions
did not extend further than the upper arm, the shoulder
remaining uninvolved. His face was natural in ex-
pression and his pupils normal throughout.

Three or four years prior to this particular fit,
J. P. was accustomed to having a series of from 270 to
280 attacks in a single day. Frequently he might be
noticed dropping on one or both knees as he was
walking across the lawn, remaining in that position
two or three seconds, regaining his feet, only to go
down again a few steps further along.

In other cases the convulsive movements may appear
in half the body, beginning in the hand or foot, while
again they may be confined to isolated groups of
muscles, especially about the face, the *parcellar epi-
lepsy* of the French.

Jacksonian epilepsy is generally due to some form
of focalized lesion in the cortex of the brain.

Jacobi * reports the case of a girl of seven years,
who began to have twitchings in the right hand four
months after an injury to her head on the left side
by falling from a swing. A few months later she had
general convulsions, which soon ceased and were fol-
lowed by more profound twitching spasms in the right
hand. At times she had "numbness and tiredness in
both arms." The majority of her attacks, which con-
tinued to occur at quite frequent intervals for five
years, were Jacksonian. Four years after the onset
of the disease, Dana noted her mental condition as
"particularly bright and mature," adding, "the
attacks were quite typical examples of Jacksonian

* "Medical Record," July 24, 1897.

Plate 8.

J. P. in a grand mal seizure, during which consciousness was partially retained. The fit is more marked on the left than on the right side, as indicated in the photograph by the prominence of the muscles in the left arm, where the contractions are tonic. The convulsion in the left leg is clonic, as shown by the blurred outline of the left knee. This patient is subject to classical attacks of Jacksonian epilepsy, affecting the left arm only. He is able to carry on a conversation while such attacks are in progress.

epilepsy. She never had any severe headaches, no vomiting, no optic neuritis; the cause probably lay in some slight degenerative changes in a limited area of the motor cortex."

The case was reported under Jacksonian Epilepsy; Adenoma of the Liver; Acute Ascites with Tubercle Bacilli. Death was due to the latter affection, symptoms of which had appeared some months previously. There was at the same time probably no connection between the various pathologic conditions and her epilepsy.

It is important to remember that true Jacksonian epilepsy rarely affects the intellect. It is essentially a disease of the motor apparatus, and from a clinical standpoint it is the simplest of recognition of all the various forms of epilepsy.

SERIAL ATTACKS.

The word serial in this connection is used in its literal sense, namely, "placed one after another; continued succession," since the convulsions in this form of epilepsy follow one another with more or less rapidity until a large number have occurred—a number ranging anywhere from five or six up to several hundred, varying greatly in different cases and in the same case at different times.

Serial attacks stand midway between isolated seizures and the severest form of epilepsy—*status epilepticus;* the gradation in this respect being: (a) single attacks which may occur daily or more often, or may be months apart, and which are not usually of great moment, so far as the patient's life is concerned; (b) serial attacks which occur hourly or oftener for days at a time, and which arc more dangcrous to life than those that occur onc at thc time; and (c) status epilepticus, in which thc attacks follow onc another with great rapidity, frequently being less than five

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minutes apart, until hundreds have occurred. The danger to life when status develops is always great.

As a rule, serial attacks most often occur in epileptics subject to *grand mal* seizures, though others, especially those who have psychic and *petit mal* seizures, may also have it.

The chief clinical difference between isolated and serial attacks lies in the point of frequency. We can easily imagine a single *grand mal* convulsion divided into four, eight, or twelve parts, each part representing a serial convulsion. As a rule, single serial attacks are not so severe as single attacks complete in themselves.

To carry the illustration a little further, we may say that serial epilepsy is a composite form of the disease, made up of many similar attacks due in all probability to the same cause, the convulsions showing themselves in the nature of repeated, sudden liberations of energy instead of a single grand discharge. It is characteristic of many cases to have three or four or more convulsions at somewhat fixed intervals, this being especially true of the epilepsies that group themselves about the menstrual period.

Some male epileptics seem to show a tendency to have more attacks at one time than at another, without any apparent cause, having a number when they do appear and remaining free from further manifestations for some time afterward, when the series will be repeated. This is one form of serial epilepsy, and it is milder in character than the serial attacks that come on at any time wholly irrespective of any periodicity and often lasting until hundreds have occurred.

Serial attacks of the latter and graver kind do not seem to show any fixed tendency to recur, differing in this way from those lighter in form.

In rare cases the temperature in serial epilepsy may reach 105° F. or over, but, as a rule, it rarely goes

Plate 9.

A good illustration of a motor epileptic. This patient had nearly 6000 epileptic seizures in seven years, the type of his disease being such that he suffered but little, if any, mental impairment at the end of that time as the result of such attacks. Conjunctival hemorrhage following a fit— right eye.

above 101° F. or 102° F. The chief distinguishing point between it and the far more fatal condition of status epilepticus is unconsciousness, or epileptic coma, which is always present sooner or later in the latter, but never in the former. The moment this condition appears, we can set the case down as status, provided the temperature is high, and the respiration and pulse frequent.

The following five cases of serial epilepsy show the duration and type of the attack, the highest temperature recorded, the total number of attacks, and the ultimate result, all the patients having recovered:

No.	Name.	Duration of Attacks.	Type of Attacks.	Highest Temperature Recorded.	Total Number of Attacks.	Result.
1	C. S.	96 hours.	G. M.	105⅘	140	Recovered.
2	O. T.	24 "	G. M.	100	11	"
3	J. K.	120 "	G. M.	101	120	"
4	T. D.	120 "	G. M.	101	40	"
5	J. P.	144 "	G. M.	100⅘	220	"

ILLUSTRATIVE CASES OF SERIAL ATTACKS.

Case I.—H. C., a man aged twenty-two, white, and single; had been epileptic since the sixth month; epilepsy was due to a cerebral palsy occurring at that age; mental condition, imbecile; he was unable to give any account of himself, nor could a satisfactory history be obtained from any other source.

Attacks, all of the *grand mal* type, occurred three or four times a week. His mental condition was worse after an attack than before; he became extremely irritable, violent, and had threatened suicide. When the attacks subsided for a few days, he became quite cheerful and good-natured, and engaged in some light employment.

On June 7, 1902, he had a severe *grand mal* attack at 5 P. M., followed by subsequent attacks as follows: 5.30 P. M., 6.25, 6.40, 7, 7.25, 8.15, 8.40, 8.45, 8.50, 9.15, when they disappeared, to be followed by one of unusual severity at one o'clock the following morning

and others at 8.30 A. M., at 3.20, 4.50, and 11.15 P. M.,
two occurring after that before five the following
morning.

His temperature after the first attack was 100° F.,
pulse 110, respiration 16, while after the tenth attack,
which occurred within four hours of the first, the tem-
perature had gone up to 101.5° F., the pulse to 118,
respiration to 20. The severest attack of the series
occurred eight hours after the first, and ran the tem-
perature up to 102° F., and the pulse to 128, after
which improvement began, with a decline in all symp-
toms. The patient was not unconscious at any time.

Case II.—C. H., a man aged twenty-three, white,
and single; had been epileptic since the fourteenth
year; epilepsy was due to trauma, the patient having
been struck on the head by a limb falling from a tree,
and causing a fracture of the frontal bone of the skull.
Mental processes were slow and dull; the mental
stupor was of a kind to indicate probability of im-
provement under proper treatment long enough
continued. The attacks at first were all of the *petit
mal* type, but changed later to include *grand mal*
also.

He began to have a series of attacks at 9.10 A. M.
on July 29, 1902, the attacks occurring thereafter
in the following order: 9.10, 9.13, 9.22, 9.25, 9.30,
9.35, 9.45, 9.52, 9.55, after which they were checked,
to recur a few hours later as follows: 1.30, 2, 2.30, 3,
3.40, 3.45, 4, 4.15, 4.20, 4.25, 4.35, 4.45 A. M.; again
stopping until 8.30 the same morning, after which
they appeared at 8.30, 9.30, 11, 12.30, 12.55, 1.50,
2.05, 2.40, and 9 P. M., the last being very mild.

The patient had thirty attacks in the series, all
told. Immediately after the first convulsion, which
was extremely severe, the temperature was 101° F.,
pulse 140, respiration 30. Three hours later the tem-
perature had reached the highest, 101.6° F., the pulse
and temperature gradually subsiding after the first
fit, all being normal after the last one.

It will be of value to bear in mind the behavior of
the pulse, respiration, and temperature in these cases
that we might compare them with their behavior under
status epilepticus.

REFLEX EPILEPSY.

A very considerable difference of opinion exists as to the propriety of designating any type of epileptic convulsion as reflex. On the other hand, many distinguished writers, including Gowers, Féré, Nothnagel, Voisin, and others refer to the *reflex causes* of epilepsy, Gowers dealing with it in this way:

"Irritation of peripheral nerves, visceral or external, is occasionally the exciting cause of convulsions which may continue as persistent epilepsy. Irritation of the gastrointestinal nerves oftener produces this effect than irritation of the nerves of the cerebrospinal system."

Gowers mentions the case of a man whose forearm was painfully bruised in an accident, and who had a convulsive attack a few days later, which began in the injured limb, with pain similar to that felt at the time of the injury. It developed later that the man suffered from cerebral syphilis, and that the injury to the arm was only the exciting cause of the convulsion.

A boy of fifteen years, mentioned by the same author, got a fragment of steel imbedded in the right cornea, causing great pain. A comrade tried to extract it with a piece of wood. As soon as the wood touched the eye, "the patient felt a peculiar thrill, the right orbicularis began to twitch, the head turned to the left, and the right limbs became convulsed; other fits followed during nine years."

A girl of nine years, who came under the writer's notice, suffered from a stricture of the urethra, which gave rise to great pain during micturition, sometimes attended with reflex convulsions. These disappeared entirely after the stricture was cut.

F. J. Campbell * reports the case of a boy who had

* "American Medicine," March 14, 1903.

convulsions for several years prior to his thirteenth
year, the attacks being infrequent at first, but occur-
ring later several times a day, all of the *grand mal*
type. He had a marked phimosis, balanitis, and
irritating smegma, the relief of which checked all
further convulsions save one that appeared the day
after the operation.

I recall a man of forty years, a mechanic of robust
constitution, unemotional and even-tempered, and
free from all pernicious habits, save that he was a
moderate drinker, who could be thrown into a severe
general convulsion by pressing with the finger-tip a
small spot, about the size of a quarter of a dollar,
near the spinal cord in the lumbar region. The first
movement of the patient would be a sharp bending
backward, when he would fall to the floor in an opis-
thotonic condition, and immediately go into a general
convulsion.

A young girl of fifteen years, whose convulsions
appeared at thirteen and who not infrequently had
200 or more in a month, had an area on the anterior
surface of the right arm about four inches long and two
wide, the spraying of which with ether would cause a
general convulsion. At first she had only three or
four attacks in twenty-four hours, but later on they
came as frequently as fifteen or twenty-five in the same
time, alternating between *grand mal* and *petit mal*.
She was well developed, muscular, and showed no
stigmata of genuine epilepsy, nor were her attacks
characteristic of the true disease.

Féré claims that "errors of refraction, necessitating
as they do efforts at accommodation, may cause epi-
lepsy," and says that it has also been ascribed to lesions
of the respiratory apparatus, traumatisms of the larynx,
and pulmonary affection, especially those of the pleura.

Nothnagel * evidently refers to the same form of the

* "Cyclopedia of the Practice of Medicine," Ziemssen, p. 257, Vol. XIV.

Plate 10.

L. B., an idiopathic epileptic, subject to both partial and general seizures which are preceded by an aura in the right arm. Irritation of the "epileptogenic zone" on the right arm, as outlined in the illustration, will induce an attack. In many partial and in some grand mal seizures consciousness is retained (Clark).

disease in what he has to say concerning "secondary epilepsy," holding this to mean that form in which "peripheral irritation, or central anatomical lesions serve as the starting-point for the development of the disease."

As a rule, the epilepsies due to such causes, *i. e.,* causes which *do not lie inherently within the structure of the brain cells,* so far as we can determine at this time, have long prodromal periods, during which the manifestations are confined to the area from which the irritation springs, and which may be either sensory, motor, or vasomotor.

Cicatrices sometimes mark· the point of development of the aura. Nothnagel says: "If there happens to be a cicatrix on the body, one may sometimes, though not always, produce a seizure by pressing upon it."

Ogles reported a case in which there was an extensive portion of the body, the upper extremity, upon the touching of which a seizure could be produced.

Great care should be exercised in such cases as this, as well as in the one we mentioned just now, in which the girl had an epileptogenous area on the upper right arm, not to confound them with hysteria. These cases bear a close analogy to the experiments made by Brown-Séquard, who was able to throw guinea-pigs into general convulsions by irritating spots on the face from which the skin had been removed.

When convulsions appear as the result of some circumscribed lesion in the brain, the prodromal period is much shorter and often entirely lacking, the attack setting in at once with a tonic or clonic spasm in a particular group of muscles, or even in a single muscle, as in the region supplied by the facial nerve or one of its branches.

Again, only the orbicularis palpebrarum, the region

supplied by the spinal accessory, or certain fingers may be involved. In these cases sensory accompaniments are almost wholly lacking, differing quite radically in this respect from the epilepsies due to peripheral irritations.

It would be interesting to pursue the study of the reflex causes of convulsions further, but enough has been said to show in a general way the type of causes that lead to such convulsions.

The essential point in it all is that the convulsions due to extrinsic causes—causes that originally lie outside of the structural composition of the cells of the brain— do not at first produce genuine or essential epilepsy, and even though I refer to them as *reflex epilepsy* I do so only in the sense that the convulsions characteristic of this and of true epilepsy alike, are essentially one, in so far as they each embrace the clinical manifestations necessary for an attack of motor epilepsy, and not because they have any parallel in etiology.

It may also be said of most convulsions which find the cause of their expression primarily and essentially in the structural composition of the nerve cells in the brain, that they have a larger motor element in them than the epilepsies which are essentially centric. So clear, indeed, is this point that it may be expressed in this way:

Epilepsies due to inherent, cytologic brain deterioration or defect show during the convulsion a minimum of altered motility and a maximum of altered mental state, while those that are due to peripheral causes show a minimum of altered mental state and a maximum of altered motility. For this reason the motor epilepsies due to peripheral irritation may exist during a patient's life, prolonged to the average extent, without much impairment of the mind.

Jacksonian epilepsy is a type of *reflex epilepsy* so far

as its clinical manifestations go, though it may be due to other causes. It is noted, however, that Jacksonian epilepsy more often runs into or is already involved with severer forms of the disease than happens in the case of reflex convulsions.

It is seldom that we find any increase in the temperature, pulse, or respiration following purely reflex convulsions, explainable, in part at least, by the undisturbed condition of the thermic centers in the brain and in part by the lessened degree of muscular involvement.

If it were practicable and desirable to make a grand division between the epilepsies due to extrinsic causes that act at first in a purely reflex manner, and the epilepsies due wholly to inherently defective conditions in the cells of the brain, it would simplify the study of an important problem to a very great extent. We might then place all the essential epilepsies in one group, the unessential in another.

EPILEPTIC EQUIVALENTS.

By epileptic equivalents is meant something that is like, that is equal to, or that in some degree takes the place of epilepsy, thus following the literal meaning of the term "equivalent," which is: "That which is regarded as equal in value, force, measure, meaning, or the like, to something else used in comparison; something that will offset, counterbalance, compensate, for the correlative indicated."

Epileptic equivalents may be referred to as fragments of epilepsy complete in themselves, since such seizures have only a partial resemblance to epilepsy in its complete forms.

Some neurologists question the existence of epileptic equivalents as independent affections, choosing rather to regard these vagrant manifestations, whether they are motor, sensory, psychic, or vasomotor in char-

acter, as indications of epileptic seizures that, for some reason, passed unobserved.

Tomlinson* is among those who reject the psychic epileptic equivalent, which up to this time has been the most commonly accepted of them all, holding as follows: "I do not believe in the so-called epileptic equivalent. I believe these states are not the equivalent of the convulsion, but they are conditions of mental aberration following the disturbance in consciousness which takes place as the result of an attack of *petit mal*, and which persists as mental status."

M. Allen Starr† describes the condition in a number of cases, referring to it as embracing a state of consciousness which differs not only from normal consciousness, but from the state of unconsciousness that usually occurs in epileptic attacks, these intermediary states being termed the *psychic epileptic equivalent*.

Starr reports several cases in illustration, and adds that "all of them appear to have 'nervous discharges' from those parts of the cortex whose activity is synchronous with the mental processes."

As previously stated, the psychic epileptic equivalent is the most common of all the equivalent states, and whether we admit or not that the disturbance in consciousness follows and is due to a minor attack of epilepsy, we know that this particular equivalent assumes two very different phases, appearing in one set of cases in the form of a sudden, maniacal outbreak of the greatest violence which lasts anywhere from a few moments to several days; while in another set of cases they appear in the form of quiet, orderly, processes of altered or lost consciousness, during which there is not the slightest indication of any motor disturbance whatever.

In cases that come under the former head we can

* "Journal of the American Medical Association," Jan. 17, 1903.
† "Familiar Forms of Nervous Diseases," 1890, p. 261.

safely assume that the *discharging lesion*, or the area in which *inhibition* is temporarily set aside, is general enough to involve the parts of the cortex that preside over all the forms of motion, together with the parts that control conscious, intellectual action.

I have previously described numerous cases of *psychic epilepsy* in which the mind was suddenly and temporarily lost, the body being in nowise disturbed in any of its parts or functions.

Some writers class attacks like these as *psychic epileptic equivalents*, a practice in which we do not agree. It seems best, so long as psychic epilepsy is a form of the disease complete in itself, not to fix on it the character of simply being "equal to or like something else."

From the number of attacks of this kind which I have had the opportunity of studying clinically in detail, I can say with quite firm assurance that they were in nowise complicated with any other epileptic state or condition, the sudden loss of consciousness appearing in all in a sharp, distinct, clear-cut manner, without the shadow of motor disturbance in any degree in any part of the body.*

When we come to the second form of the so-called *psychic epileptic equivalent*, that in which there is motor disorder, with prolonged, intense violence, we have an entirely different proposition, for here such disturbance often follows an epileptic seizure of some notable kind.

So closely interwoven is the fit itself with the sub-conscious period of motor violence that follows, that the patient himself cannot distinguish between the fit and the subsequent state. The two are dovetailed into such close apposition that the line of demarcation is destroyed by merging as much into one side as in

* "The Silent Forms of Epilepsy," by William P. Spratling, "New York Medical Journal," Oct. 11, 1902.

the other.   Nor is it possible to say absolutely whether
mental and physical violence ever occurs indepen-
dently of a forerunner in the form of a very mild
seizure; but it seems reasonable to suppose that such
may be the case, for this violent state is closely analo-
gous and often identical with ordinary outbursts of
mania not associated with epilepsy in any form.

Such mental disturbances appear as strange, violent
impulses of short duration.   The patient may begin to
speak suddenly in an unusual and incoherent way, or
exhibit rude behavior, or to destroy various objects,
while others in this state seem to possess superhuman
strength and not infrequently wound or kill those
about them.   Others lose all sense of decency and
are given to exposing their person in public places.
Lésague has shown that those among the insane who
practise indecent exposure are often epileptic, a fact
the author had ample occasion to verify in his care
of some two hundred insane epileptics for a number
of years.

These unprovoked assaults and indecencies are
usually committed while the individual is unconscious,
and he knows nothing of it until told of it afterward,
when he usually falls into great depression or has a
regular convulsion which breaks up the abnormal
state.

Such cases as these may properly be called *psychic
epileptic equivalents*, for they are manifestly inter-
changes for fits of ordinary character.

More frequently, however, the equivalent state that
follows is only a part of the result of a mild seizure
that could not be differentiated in point of time from
the equivalent itself.

The case of R. F. H., mentioned on p. 173, very
clearly illustrates the psychic epileptic equivalent
state when that state is dependent on seizures of
another kind.

The following is from the author's report * of this case: "The patient, a man of forty, an epileptic for eighteen years, entered the Colony early in December, 1901.

"His family history was not good, while the patient himself drank and smoked to excess until a year after his epilepsy developed, at the age of twenty-one. He is a man of intelligence and was profitably employed as a commercial traveler.

"It has been his habit for years to keep a diary, noting therein his epileptic seizures with a certain Greek sign, to conceal the identity of his trouble as far as possible.

"He left Rochester, N. Y., September 26, 1901, for a prolonged Western trip, and his diary faithfully records the activities of each day up to December 14th following, when he entered the Colony. The notes in the diary are in detail, giving the names of the places visited; the railroads he traveled over to reach them; the hours of arrival and departure; the names of the firms called on; the result of these calls; together with frequent notes to show how he felt, how many letters he wrote, and many other things of a personal nature. We will omit these diary notes between September 26th and November 15th, and give them from that time on as copied from his diary by the patient himself:

Nov. 15. Little business to-day. Went to Kalamazoo in afternoon. Wrote two letters. Serious epileptic collapse on the street this morning; at Elkhart.

Nov. 16. Saw Kalamazoo trade. Slight touch of epilepsy in evening. Wrote three letters.

Nov. 17. Sunday. Spent the day indoors, writing and figuring. Wrote three letters. Epileptic attack, after a smoke this afternoon.

* "Journal of Nervous and Mental Diseases," August, 1902: "Epilepsy in its Relation to Crime," by William P. Spratling.

Nov. 18. Visited trade in Battle Creek, Marshall, and Albion. Wrote three letters.

Nov. 19. Did the Jackson and Hillsdale trade to-day. Not much business. Had attacks of epilepsy both forenoon and afternoon.

Nov. 20. Visited Angola and Auburn. Slept at Columbia City. Wrote one letter. Had epileptic attacks both forenoon and afternoon to-day.

Nov. 21. Columbia City and Fort Wayne both attended to-day. Toledo by night. One slight epileptic attack to-day. Wrote four letters.

Nov. 22. Spent the day in Toledo. Up to Detroit on evening train. Received my long-looked-for "medicine quantity" letter at last. Wrote four letters.

Nov. 23. All day in Detroit. Did little business. Troubled with chills and fever. Took strong dose of quinine at bedtime, also some whiskey.

Nov. 24. Spent all day on correspondence and literature, in hotel. Wrote four letters.

Nov. 25. My diary says: total epileptic collapse.

Nov. 26. My diary says I was at Pontiac, also Detroit, but further than this I know nothing, and have no recollection whatever regarding the day.

Nov. 27. My diary says I got up at 8.30 by my watch, i. e., railroad time. Utterly worn out. No memory left. Saw a doctor. Am to be sent home to-night on M. C. R. R. No. 3 lower berth on sleeper. Night message sent to my home doctor, my firm, and my landlord. (I remember none of this.)

Nov. 28. My diary says: Breakfast on train. It also records $30.00 turned over to my employer. (I have no personal recollection of these items.)

Nov. 29, 30, and Dec. 1. At hospital. (No record or recollection of this.)

Dec. 2. My diary says: Up and dressed after dinner. My employer called. I am weak but mentally clear to-day. No recollection hardly.

Dec. 3. My diary says: Up at 9 A. M. No further record.

Dec. 4. My diary says: "Up at 9 A. M. Dressed fully. (From this I infer that I only used my dressing-gown on the other two days.) My employer called

with the Craig Colony papers, which the doctor signed later.

Dec. 5. Up for breakfast. Back to bed. Up again 10 A. M. Wrote a letter. Had an epileptic attack at 7 P. M. Chewed a thermometer when being put in my mouth to take temperature. (I have a sort of hazy recollection of this—spitting out cut glass, etc., but nothing definite.)

Dec. 6. Up and dressed for breakfast. Paid $1.25 for thermometer. To-day's visitors were W. H., Mrs. H., and Dr. J. Wrote one letter.

Dec. 7. Up and dressed 7.30 A. M. Got shaved. Went outside and over home. Saw Mrs. H. Back to hospital for dinner. Van called 8 P. M.

Dec. 8. Sunday. Three ladies, one man called on me to-day.

Dec. 9. Depressed. Wrote two letters. No further record in diary.

Dec. 10. Diary says: Epilepsy between 4 and 5 P. M. Chewing severe. Record of four letters written. (Beyond these facts diary does not throw any light, nor does memory recall the day at all.)

Dec. 11, 12 and 13. Back home again. (No recollection of these three days.) Diary records two letters received and one written, but memory fails to recall them. And these were vitally important days, too, when I was making arrangements for my sojourn at Craig Colony, and trying, presumably, to straighten out my affairs before leaving. I discovered, on my return for a week in February, that I had given away a number of personal effects to friends, and my failure to recall this fact caused a few rather embarrassing blunders. For instance: I calmly arranged to have some furniture taken up to the Colony, which I had previously sold to my landlord.

Dec. 14. My employer accompanies me to Sonyea. Diary makes note of the main facts, but my memory fails to respond when I try to recall them now.

Dec. 23. From the 14th till now—a week—my diary makes note of letters received and answered. Also states that on Tuesday, December 17th, I was epileptic all day, and on the 18th there is also the sign of epilepsy, but as far as personal recollection goes,

my memory is *nil*.   I have a sort of "hazy," "dream-like" recollection of matters during that period, when I try to recall it by going to my correspondence and such like, but am unable to fix things at all definitely. (I have carbon-manifold copies of the letters, which show them to be perfectly normal and sensible, yet my memory fails to retain the incidents referred to in said letters.)

From December 23d till to-day—over two months— I have been in better health than for years past, and have had but three epileptic attacks.   (None of them was serious, and each clearly traceable to exciting or depressing primary causes.)

The main features noticeable in this somewhat disjointed description of my goings-on from September 26th till to-day, and which I desire to emphasize, is the practical loss of memory from November 15th till December 23d.   While my diary keeps a fair record of most of the time, and my mind recalls certain days and incidents dimly, I really seem to know nothing as to my existence during that period, and might as well have been in a comatose condition.   Though my letters and comments seem perfectly lucid and rational, I have no recollection of most of the incidents there narrated.

"It appears from the foregoing that he suffered a 'serious epileptic collapse' on November 15th, and had other isolated attacks daily up to the 20th, when they ceased, only to reappear again in the form of 'total epileptic collapse' on the 25th, from which time until December 23d, a period of twenty-eight days, he had a 'practical loss of memory.'

"It does not appear that unconsciousness during these twenty-eight days was absolute and complete; for the patient himself says, with every evidence of honesty and sincerity and without any motive for evasion, that while his mind could recall 'certain days and incidents dimly,' he really seemed to 'know nothing' of his existence during that period.

"One of the most remarkable features of the case

was the patient's seeming ability to write down certain incidents and impressions acquired while he was in a subconscious state, and yet have no knowledge of the fact later, only knowing that he had done so by seeing his own writing on the pages of his diary, or copies of letters he has kept.''

States in epilepsy in which the individual acts without volition—acts in a purely automatic way—are of great interest to the medical jurist and constitute one of the gravest medicolegal problems we have to encounter. These states are more fully dealt with in the chapter devoted to the medicolegal aspect of the disease.

OTHER EPILEPTIC EQUIVALENT STATES.

In addition to the equivalent states described above, others occur of a sensory, sensorial, or motor character, their connection with epilepsy being demonstrated by their coexistence in the same person, or in the patient's family, and by their essentially paroxysmal nature. Some of these may be regarded as limited or incomplete seizures, as, for instance, those that partake of the nature of a definite aura which fails to lead up to a full convulsion.

Among the visceral epileptic equivalents may be mentioned *angina pectoris*, which may precede, coincide with, or follow a regular attack of epilepsy. According to Féré, when the convulsive seizure begins in such cases with a violent pain in the head, extending to the neck and precordial region, it is impossible to mistake the implication of angina pectoris. In some cases the entire attack may be limited to this painful aura.

Paroxysmal tachycardia has been thought to have some relationship with epilepsy, but the fact that patients in such cases are usually free from any other evidences of an epileptic nature, makes the assumption

doubtful at least. In such cases convulsive move-
ments as well as the loss or disturbance of conscious-
ness are both lacking, without one or the other of
which in some degree epilepsy in any form cannot
be present.

Féré reported a case in which he asserted *unpro-
voked laughter* to be an equivalent of epilepsy. In
the absence of any of the distinctive phenomena that
accompany epilepsy, we should here again hesitate to
accept such a thing as an epileptic equivalent, espe-
cially when we recall the association at times of uncon-
trollable laughter with hysteria.

Bombarda * declares that certain *sleep states*, such
as nightmare, starting in sleep, pavor nocturnus, or
night terrors, erotic dreams, and seminal emissions,
are simply equivalents of *petit mal epilepsy*.

Hamilton and Zuccarelli agree with Bombarda in
believing *nocturnal pollution* to be a true attack, but
from such views conservative neurologists very prop-
erly dissent.

Among other forms of epileptic equivalents are
asthmatic affections, especially when the asthma sud-
denly attacks persons previously in good health.
Féré is authority for the statement that the "existence
of asthma with psychic disturbance, hallucinations,
terrors, fear of death, false croup, and spasm of the
glottis are also included among the convulsive phe-
nomena assumed to be allied to the epileptic state."

Some writers hold the symptomatic analogy between
spasm of the glottis and incomplete epileptic seizures
to be very great; among them being Rilliet and Bar-
thez, who describe the condition in substance as fol-
lows: "The infant is seized suddenly with an attack
of convulsive suffocation. Respiration is suspended,
the face dark and congested, the head drawn back-
ward, the mouth wide open. The child is restless and

* "Journal of Nervous and Mental Diseases," Vol. XXVII, p. 342.

carries its hand to its neck as if to remove the obstacle to respiration; then after a few seconds of this dyspnea it makes several short, sharp inspirations, without intervening expirations, soon followed by a faint and wailing inspiration, or sometimes by a convulsive, noisy, and jerky expiration; at the same time the extremities stiffen, the thumb is carried into the palm of the hand, the fingers stretched on the metacarpus. . . . . Involuntary evacuations take place. Most frequently consciousness is preserved; sometimes it is lost."

J. Lewis Smith,* Holt,† Strumpell,‡ and others fully describe the same condition, all agreeing that attacks of laryngismus stridulus, or spasm of the glottis, may cause convulsions and unconsciousness. J. Lewis Smith refers to the affection as "internal convulsions," ascribing to it clonic and tonic contractions of the respiratory muscles most frequently, but occasionally those of the face and limbs also occur.

Trousseau says internal convulsions consist principally in a *spasm of the diaphragm* and of the muscles of the abdomen and chest; but it appears that the muscles pertaining to the larynx are also affected at the same time with these.

It is the opinion of writers generally that a neurotic element underlies the affection in most cases.

It has already been stated that epilepsy is sometimes caused by *pertussis*, and attention is called to the fact that the seat of local irritation, the convulsive closing of the glottis, is similar in nature and produces similar results. I do not for a moment class spasms of the glottis with true epilepsy, but with the so-called epileptic equivalent. The seat of the irritation in such cases is problematical. The brain may be affected in

* "Diseases of Children," 1896, p. 634.
† "Diseases of Infancy and Childhood," 1900, p. 672.
‡ Strumpell, "Textbook of Medicine," 1892, p. 142.

a reflex manner only. The pathology of the condition is in obscurity at this time.

It is unreasonable to suppose that true epilepsy often, if ever, follows this form of so-called epileptic equivalent. If it did, epilepsy would be vastly more frequent than it is now. Personally, I fail to recall a single instance in which this was the case.

Among the prominent sensory equivalents may be mentioned the *gastralgias*, *enteralgias*, *nausea*, and *vomiting* when they suddenly occur in persons known to be free from diseases of the gastrointestinal system, some of which doubtless constitute incomplete forms of gastric auræ, while certain hunger states, to be distinguished from simple bulimia—or desire for food— have been associated with epilepsy.

Féré * has reported several cases of the *hunger evil* in which the abnormal hunger state appeared both as an aura in classic attacks of epilepsy and as an equivalent in others.

The *migraines* also belong to this group. The relationship between these affections and epilepsy has long been in dispute. I believe it is associated with the disease, especially in women, who more frequently show a periodicity in convulsive phenomena than men. Unquestionably some of the lighter forms of epilepsy pass for periodic sick headaches. It is the rule for psychic seizures to be followed by an intense, protracted pain in the head, that may persist for several days.

Moreau de Tours states that the migraines of epileptics leave behind a profound stupor, often coming on and disappearing with the suddenness of epileptic paroxysms. In other cases a periodic migraine seems to replace a periodic epilepsy.

Tissot, Parriot, and Lieving are among those who admit the connection between migraine and epilepsy.

* "Revue de médecine," July 10, 1899.

Before we can understand the relationship between periodic migraine and some of the lighter epileptic states or equivalents, we must have a thorough understanding of the scope, character, and causes of such states and equivalents.

PARTIAL EPILEPSY.
(Synonymous with Jacksonian Epilepsy.)

We find occasional references in the literature to the *épilepsie partielle* of the French writers, considerable distinction being given it both by Voisin and by Féré.

Descriptively speaking, partial epilepsy refers to that form in which the convulsions are limited to half of the body, to one arm or leg, or to a single group of muscles, the latter, as previously stated, being termed by the French *parcellar epilepsy* (*Épilepsie parcellaire*). It was first described by Prichard in 1822, being next referred to by Bravais in 1827 under *hemiplegic epilepsy*, and later still, in 1831, by Elliottson under *partial epilepsy*.

The studies and descriptions of all these writers, however, were inconclusive and incomplete as compared with those made by Hughlings-Jackson, begun in 1866, and which were of especial value on account of the work of this distinguished physician in studying the malady from the diagnostic point of cerebral lesions.

At the outset we must guard against the mistake of confusing the *incomplete* attacks of ordinary epilepsy with the *partial* forms of the disease, the latter being a distinct type in its own manifestations; the former, an ordinary attack from which some of the important symptoms are missing, this form more nearly resembling the epileptic equivalent already described.

The causes of partial epilepsy are numerous and may be general in some degree, or purely local, com-

prising, under general causes, meningitis, acute or chronic, syphilitic infection, uremic poisoning, exaggerated emotional states, alcoholic excesses, and trauma—especially of the skull with injury to the brain, cerebral hemorrhage, thrombus or embolism, and extrinsic causes that act in a reflex manner, like injuries to peripheral nerves, old cicatrices, and various forms of irritation in the different viscera.

As a rule, there is no cry in an attack of partial epilepsy, while consciousness during the attack is seldom completely lost, the patient being able in some instances to witness his own seizure and even to converse while it is present. Nor are such attacks usually preceded by an aura, though in some cases there may be sensations of numbness, tingling, pain, or coldness in the extremities or in certain localities, not differing essentially in this respect from the aura which precede regular epilepsy. Almost invariably the spasm begins in the face or in an extremity and progresses according to certain fixed rules described by Hughlings-Jackson.

When the spasm begins in the face it most frequently affects the eye, or the mouth, the former moving laterally or upward at an angle, with twitching of the eyelids. The mouth is drawn upward and to one side, the spasm radiating to the rest of the face on the affected side, affecting the muscles of the nose, the alæ nasi especially, causing that organ to draw up in a way that produces a grinning aspect—a condition to which Voisin gives the name "rabbit's nose."

At times the teeth are gnashed together. The muscles of the neck are next involved, the head turning to the side to which the mouth and eyes deviate. Any further extension of the spasm affects the superior portion of the body, then the inferior portion on one side, and when it is the right, there is apt to be aphasia.

Again, the spasm may begin in the thumb or one

of the fingers, or two or three of them may be simultaneously involved; the next step is the flexing of the fingers in the palm, over the thumbs, as a rule. Then it spreads to the forearm, the elbow, and the upper arm, stopping short in some cases before involving the shoulder-joint; while again it may proceed, involving this, and then the head, finally affecting the lower extremity, making the involvement of half of the body complete.

When it starts in the lower extremity, it generally starts in the great toe, which is extended or flexed convulsively, then travels up the leg to the trunk. In movements in the upper extremity extension predominates, while in those of the lower extremity, flexion is more often noted.

We may note a tonic phase of the spasm only in any of the parts affected described above, or they may be followed by clonic movements also; the loss of consciousness that follows the latter in some instances is short and the coma of brief duration.

No matter how mild the attacks in either degree may be, whether they are tonic only or are clonic also, certain physical results are nearly always noted afterward, consisting of partial or complete temporary loss of power in the parts involved in the spasm. For instance, the face, when drawn strongly to one side during the spasm, may remain in an unbalanced position for several days, the length of time depending upon the duration and intensity of the original discharging lesion in the brain. Series of attacks are especially apt to cause local exhaustion paralysis, the same condition resulting when the leg, or arm, or any particular group of muscles, is implicated in the primary spasm.

Fixity in locality is a characteristic of partial epilepsy, its symptoms, as a rule, being repeated with each seizure, in this respect, in exact duplication with

wonderful regularity, though the degree of intensity may at times vary greatly.

Partial epilepsy in rare instances may be replaced by general tetanoid shocks that are attended with general excitement, indicating a more universal involvement of the brain, particularly disturbance in the portions that underlie the psychic life. Sometimes, too, such attacks are replaced by marked general trembling which may persist for a day or so, the patient being meanwhile in an uneasy, apprehensive mental state.

It is only in those forms of motor spasms or disturbances in which the impairment is local that the mental faculties remain unaltered.

Charcot described two special forms of hemiplegic partial epilepsy, tonic contractions being characteristic of one, the other having vibratory movements which affected the contractured limbs during the tonic phase, followed by regular clonic movements later on.

Infantile hemiplegic epilepsy sometimes differs from partial epilepsy in that the hemiplegia follows a series of convulsions early in infancy, the convulsions antedating the hemiplegic state, though the two forms are not infrequently present in the same individual.

Féré mentions ophthalmic migraine as constituting a partial sensory epilepsy that begins with excitement, violent pains, scintillating scotomata, etc., the pain radiating over the side of the cranium corresponding to the affected eye and being accompanied by nausea and vomiting. As a rule, visual disturbances manifest themselves first in the way of dimness of vision or hemianopsia, while in others blindness may be complete.

Scintillating scotomata are held to be of special importance, generally occupying the peripheral portion of the field of vision and consisting of "larger or smaller balls of fire, perhaps in the shape of a toothed

wheel of red, white, or phosphorescent aspect, and in rapid vibratory motion, or rotating around its center.''

The patient is confused at first by these extraordinary phenomena and cannot give a clear account of them, but is able to do so later. That they are dependent upon centric causes is shown by the fact that they continue present when the eye is closed or when the patient is in complete darkness.

Occasionally the scotomata consist of simple, luminous, zigzag lines and figures resembling an electric spark.

Sir Lauder Brunton * discusses at length the possible relationship between migraine and epilepsy, saying in part: "If the terminal branches of the temporosphenoidal artery become contracted like a bit of piano wire, as the one which runs up my forehead does during a headache, the nutrition for the center of sight in the brain must necessarily be impaired, and if the spasm should extend further down the artery, the centers for hearing, taste, and smell will also suffer.

I think it probable that such impairment is the cause of the indistinct vision in hemianopsia, *i. e.*, blindness to all objects on one side of the body, either to right or left, even of complete blindness and of zigzags which occur either before or during an attack of migraine.''

This distinguished author says, moreover, that, while the idea to some may be far-fetched, he inclines to believe that the fairies which many people declare they see are nothing more than the colored zigzags of migraine modified by imagination, and in some cases occasioned by an abnormal condition of one or the other eye.

Hallucinations of sight during epileptic attacks in which there is mental disturbance are not uncommon,

* "Hallucinations and Allied Mental Phenomena," "Journal of Mental Science," April, 1902, p. 227.

but, in my experience, such ocular manifestations as those described by Féré and alluded to by Brunton, in which this condition is the only indication of an attack of epilepsy, are exceedingly rare, while the very fact that visual aura are so common before ordinary attacks, and often so complicated in their formation, constitutes additional reasons for the existence of partial epileptic attacks that find the eye in ophthalmic migraine the center of disturbance.

In such conditions the pupils are usually contracted, while in ordinary epilepsy they are the reverse.

At the French Congress of Alienists and Neurologists at Nancy, August, 1896, Raymond and Sougues reported a case of partial epilepsy in a man fifty-four years old, of three years' standing, the man suffering from acromegaly. The epileptic attacks were typically Jacksonian, limited to the right arm and right side of the face. It was stated that the hypertrophy of the pituitary gland present in acromegaly constituted a tumor capable of exciting, from a distance, the cortical psychomotor centers.

TETANOID EPILEPSY.

Attention was first called to this form of epilepsy by Prichard* in 1822, and it is believed to be exceedingly rare, a fact which may be explained, in part at least, by the failure of students of epilepsy to give it a place of its own in epileptic literature.

From a consideration of the patho-physiologic changes that occur in the brain during an epileptic seizure, it is difficult to understand why there should be two distinct forms of spasm during the progress of a fit; why the tonic contraction alone does not suffice for the complete liberation of nervous energy.

Apparently this is not the case, for, except in the rarest instances, we meet with two forms of motor

* "Diseases of the Nervous System," p. 108.

disturbance, tonic and clonic, neither of them being fixed nor invariable in degree, both differing greatly in different cases, and even in the same case at different seizures.

In the sense in which we use *tetanoid* in this connection, it is practically synonymous with tonic; tetanoid epilepsy, or tetanoid seizures in epilepsy, as it is best to call it, consists in tetanic or tonic-like contractions that occur wholly independent of any clonic movements whatever. Care must be taken not to confound such seizures with tetanus in any form, for they have no connection in any sense other than that conveyed by the brief disease-picture we look on when the tetanic state is present—momentarily so in epilepsy; more continuously so in true tetanus.

Prichard divided tetanoid epilepsy into two forms, convulsive and tetanic, speaking of the latter as follows: "The less frequent or tetanic form is distinguished by sudden fits of coma, or loss of sense and consciousness, without convulsions, but attended with a tonic spasm of the voluntary muscles, the whole trunk becoming during the fit rigid and inflexible."

In speaking of the clonic form—the convulsive—he described it as being similar in some respects to ordinary epilepsy, with clonic convulsions: "The patient is suddenly seized, the limbs stretched out, the whole trunk extended, and fixed in rigid spasm; eyes wide open and staring; pupils strongly contracted; the convulsive and tetanoid forms are closely allied, and both may attack the patient within a few hours."

Delasiauve, Jones, Grasset, Gowers, and Féré allude to the affection in some way, the former appearing to think that tonic and clonic epilepsy cannot occur as independent affections.

Jones speaks of "a very perilous and fortunately very rare" form of tonic contraction that holds the muscles of the chest in a state of rigidity long enough

to cause death. A case of the kind came under my own observation, that of a woman of thirty years, who was subject to *grand mal* attacks, but she had never previously been known to have a convulsion like the one about to be mentioned.

She went to a faucet over a marble basin to get a drink of water before leaving the building, with another person. After she had been gone five or six minutes, she was called, but did not respond, and a moment later was found standing by the wash-basin with hands lying flat on the marble surface, her feet a little apart, the head drawn well forward and down, dead. The entire muscular system was as rigid as wood and remained so until after the patient was laid on the floor. The face, neck, and upper part of the body showed the usual dark discoloration that follows death from asphyxiation. It was clearly evident that she had suffered a powerful tetanic convulsion involving the whole body, fixing in a vice-like grip the muscles of the chest, causing death by the forcible *locking up* of the respiratory movements.

When the body became limp, it did so without preliminary tremors or twitching of any kind, the wood-like hardness of the muscles almost instantly melting away.

I have known of other cases in which death occurred during the attack, with the patient in a different attitude, and was due probably to the same cause.

Gowers,* while stating that most attacks of epilepsy consist of both tonic and clonic spasms, says that in some attacks one or the other of these may be missing; tonic spasms, light in form, being the commoner of the single type kind. "A patient falls unconscious, is rigid a few moments, then is better. Occasionally, more severe attacks consist of only tonic spasms, as is the case of a boy whose fits were as follows: His

* "Epilepsy and Other Convulsive Diseases," 1901, p. 95.

head was first turned to the right, then his arms became extended and rigid, the right being more abducted from the body than the left. Both elbow-joints were fixed and his fingers were flexed in the interosseous position. In a few moments the spasms ceased, lasting a little longer in the hands than elsewhere. There was no clonic spasm.''

A girl of seven, of delicate physique, an adopted child whose ancestry was obscure, was entirely well up to three years and a half. She appeared ill one day, listless, dull, and pale, and had a loss of appetite. All of these symptoms became more pronounced during twenty-four hours, when nausea and vomiting set in, followed by convulsions. The convulsive movements consisted of *a stiffening out*, the entire body being rigid and firm, the head drawn strongly back, the back powerfully arched in a typical opisthotonic position. The legs and arms were straight. After the spasm had lasted from thirty to forty seconds, it passed away almost instantly, and at once the little patient was in possession of all her faculties, though she felt physically weak for some hours afterward. A month later a similar attack occurred that shaded off into clonic spasms, and during the following three years and a half she had from two to five attacks a week, some tonic only, some clonic during the latter stage.

She also had what her friends called "nodding spells." While at play she would suddenly stop and vigorously nod her head, the chin falling on the chest. She would at once straighten up, seem a little confused and languid, but go on with her play. When this nodding spasm occurred while she was at the table, her forehead would strike the table with great force, at times breaking the plate she was eating from. I saw her once just after her head had vigorously struck a dish of oatmeal she was eating.

After two or three seizures had occurred in a day, as occasionally happened, she would suffer an exhaustion paralysis involving the tongue and lips. She could not speak without great difficulty and indistinct mumbling of words, and suffered a constant drooling, the lips appearing full, tense and flaccid, reminding one of the thick, expressionless lips so often seen in idiocy and other inferior mental states.

L. Pierce Clark * reported the case of a male, twenty years of age, of good muscular physique, save for a slight paretic condition of the right side which followed a hemiplegia at the fourth or fifth year, who was subject to series of tetanic spasms which were occasionally followed by classic attacks of *grand mal*.

During the tetanoid seizures all muscles were in a state of rigidity from twenty to thirty seconds, the back being well arched each time. The patient could be lifted bodily from the floor by supports under the heels and head. He finally had a series of such attacks which ended in status, and caused his death.

The nodding spasms referred to in the case of the girl are known as *salutatory spasms* or *spasmus nutans*, and are mainly found in children.

MYOCLONUS-EPILEPSY.

Myoclonus-epilepsy is an association disease of a comparatively rare type. It is characterized by paroxysmal asynchronous, bilateral, lightning-like contractions of the trunk and of the proximal muscles of the extremities with varying intervals of entire freedom from such movements, and accompanied by a more or less persistent *grand mal* type of epilepsy.

The condition was first carefully described by Unverricht in 1891 under the title of Family Myoclonus. In about a fourth of the cases it occurs sporadically. In a monographic study of the condition, Clark

* "American Journal of Insanity," Vol. LV, p. 583.

and Prout * report 57 cases of myoclonus-epilepsy, which number includes four of their own from the Craig Colony. Since their study, several additional cases have been reported by Sepelli, Lundborg, and others. One is led to infer, however, that the cases are by no means so rare as unrecognized, cases being diagnosticated as choreic epilepsy, epilepsy with multiple tics, or, perhaps, epilepsy associated with hysterical tics.

The hereditary factors of the association disease are even more pronounced than those of true idiopathic epilepsy, all showing an intense neuropathic stock. The immediate excitant of the disease is the minimum of importance. Of all the alleged causes, some form of mental stress takes first place, but this cause is often absent.

In the development of myoclonus-epilepsy, which usually occurs in early adolescence, epilepsy develops first in about half of the cases; in a third the two diseases have a simultaneous onset. The seizures are usually more or less imperfect *grand mal* in type and are generally preceded by increased myoclonic contractions. Commonly, the seizures are followed by free periods of contraction for hours or days. The myoclonus portion of the association disease is more frequently atypical than the epilepsy; the hands, feet, and face are often affected, and the paroxysmal character of periodic contractions is less sharply outlined. The diaphragmatic or wood-chopper's grunt is a later addition to the myoclonic symptoms, indicating an involvement of this muscle.

The diagnosis of typical cases is easy. Errors are generally due to laying too much stress on single symptoms of the disease. The lightning-like contrac-

* "The Nature and Pathology of Myoclonus-epilepsy," "American Journal of Insanity," Vol. LIX, No. 2, October, 1902.

tions of the trunk and the proximal muscles of the extremities, which are asynchronous yet bilateral, and which are not possible of production by the will, occurring in an epileptic should enable one to make the diagnosis.

The prognosis as to recovery is bad. Life is usually curtailed more than in simple epilepsy. Death occurs commonly before the climacteric from emaciation and general marasmus or status myoclonus. The pathology of the affection is not yet definitely determined. Autopsies in several cases have failed to show gross macroscopic changes sufficient to explain the condition. However, from recent researches, it is quite certain that its pathogenesis does not rest either in the muscles or the spinal cord, although these may and often do show secondary changes. The association disease, as well as simple myoclonus, is probably cerebral in origin. Its type of cortical degeneration should be somewhat analogous to that of paralysis agitans, chronic and senile chorea, and the like chronic convulsive disorders which develop upon a soil of degeneracy, plus some sort of autointoxication, possibly thyroidal in origin, as Lundborg has recently urged. As to the histocytologic changes in the cortex, the conclusions of Clark and Prout, who carefully studied it in one case under ideal conditions, are worthy of attention, inasmuch as they form a material basis to the conclusions of Raymond, Ribot, Didé, Venga, and Gonzales, and others who state that its real pathology is cortical. They believe that the lesion of myoclonus-epilepsy is in the cerebral cortex, involving the nucleus and the intranuclear network of cells of both sensory and motor types. Its pathogenesis appears to be an intoxication or autointoxication of these cortical cells, probably brought about by a faulty chemotaxis of these same cells because of an inherent organic anomaly. While the epilepsy and

the myoclonus maintain their separate morbid entity, the two are closely allied, and indeed are often found as indissolubly associated clinically and pathologically as are the motor and sensory functions of the cells they involve.

FREQUENCY AND TIME OF EPILEPTIC SEIZURES.

Epileptic attacks occur in point of frequency anywhere from once a year or less often up to several hundred a day. The greatest number of well-defined attacks in one hour that have come under my observation in one day was 280; another patient had 519 in forty-nine hours, dying at the end of that time in status epilepticus. The convulsions in this case were general; in the former they were mostly Jacksonian.

J. P. in seven years had attacks as follows:

Year.	Day.	Night.	Total.
1896	236	154	390
1897	538	641	1179
1898	465	517	982
1899	578	633	1211
1900	367	533	900
1901	275	281	556
1902	302	306	608
Total			5826

E. C. in five years had 26,124 *petit mal* and psychic attacks in alternation, which failed to impair her mental faculties in any respect.

Year.	Day.	Night.	Total.
1898	2784	18	2802
1899	6239	157	6396
1900	7145	13	7158
1901	8878	717	9595
1902	173		173
Total			26,124

In 1374 cases under my observation the approximate frequency of the attacks was as follows:

13

Weekly or more often.................... in 775—56 per cent.
Every two weeks....................... " 173—12⅓   "
Every three weeks .... "   61— 4½   "
Every four weeks .... " 244—17½   "
Every eight weeks..................... "   42— 3   "
Every twelve weeks.................... "   49— 3½   "
Every six months or over.............. "   30— 2¼   "

It is understood that these frequency periods are approximate only. Few cases, save those in the four-weeks period—most of which are in women—show any fixed periodicity. The greater number in the four-weeks period is accounted for by the stress incident to the menstrual flow. Among those having attacks weekly or more often, half or more have attacks daily or several times a day.

The following diagram shows the number of seizures by hours at the Craig Colony during the year 1902. The summary includes 100,296 seizures; 41,423 in females, 58,873 in males.

Hour of attack.	Number of attacks.	
1 A. M.	3793	
2 A. M.	5017	
3 A. M.	5746	
4 A. M.	4652	
5 A. M.	3712	
6 A. M.	4264	
7 A. M.	4258	
8 A. M.	4581	
9 A. M.	4413	
10 A. M.	4420	
11 A. M.	4562	
12 Noon	4082	
1 P. M.	4120	
2 P. M.	4132	
3 P. M.	4189	
4 P. M.	3763	
5 P. M.	3594	
6 P. M.	3865	
7 P. M.	3256	
8 P. M.	3771	
9 P. M.	4649	
10 P. M.	4184	
11 P. M.	3935	
12 Mdnt.	3307	

CHAPTER VII.

STATUS EPILEPTICUS.

BY L. PIERCE CLARK, M.D.

Synonyms: État de mal; Acute Epilepsy.

Historic.—The first scientific study of status epilepticus was begun in the early part of the ~~twentieth~~ 17ᵗʰ century at the Salpêtrière and Bicêtre Hospitals of Paris. It was called "*état de mal*" by Bouchet and Cazauvielh in their work upon the condition in 1825. Bourneville's name, however, is most deservedly connected with our present knowledge of the subject, as his works cover a period of the past thirty years.

While some think the condition of status to be rare (largely found in asylums), most epileptologists believe it is one of the chief factors in mortality statistics of epileptics both in and outside special institutions for this class. Status epilepticus is the true climax of epilepsy; that is, an epileptic is foredoomed to die of the status as the maximum development of the disease, if all possible complications such as intercurrent affections are removed. Although there are notes of at least 300 or 400 cases of the status in medical literature in which fairly accurate data of a clinical nature may be obtained, yet careful and complete clinico-pathologic studies of even a few cases are comparatively rare.

Definition.—As to what constitutes status, authorities still differ, and it is still without exact definition. For typical cases, however, the condition may be defined as follows: *Status epilepticus is the maximum development of epilepsy in which one paroxysm follows*

another so closely that the coma and exhaustion are continuous between seizures. It is sooner or later attended by a marked rise in temperature, pulse, and respiratory frequency. The latter accompaniments are the indices to the degree of exhaustion and its fatality.

As status is confined largely to the *grand mal* form of epilepsy its variations are obviously less bizarre in their exhibition than epilepsy proper. However, in rare instances it may be composed of equivalents such as delirium, stupor or coma, cough or hiccough, and a variety of psychic states which have for their bases cortical discharges resulting in more or less complete physical and psychic exhaustion. From the convulsive standpoint, however, status is usually composed of ordinary *grand mal* seizures which preserve their individuality or become imbricated tonic or clonic spasms. The seizures may be lacking in tonic or clonic elements, but the former is the one usually curtailed if either is omitted. Status periods composed entirely of tetanoid or statuesque attacks are not unknown (Clark). Bourneville has attempted to classify true status epilepticus into three or four different varieties, and serial periods into two classes, but a real difference in types of status does not exist, and serial attacks are but stepping-stones to status, depending much upon the nature of treatment employed whether or not they may end in true status.

Forms of Epilepsy in which Status Occurs.—It usually appears in those cases of idiopathic epilepsy in which *grand mal* seizures predominate. It may occur in psychic epilepsy.* Fatal status, however,

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* I desire to call especial attention to the form of status that follows psychic seizures. It is attended by a few light convulsive movements only. often so light as to almost pass unobserved, the patient quickly sinking into coma with increasing temperature elevation until death supervenes, often within a few hours. It is interesting chiefly on account of the gravity of the condition as compared with the apparent insignificance of the convulsions that led to its creation. (Spratling.)

is most often recruited from the *grand mal* type. No definite rule can be laid down governing the occurrence of status in any particular case of simple idiopathic *grand mal* epilepsy, with the exception that those liable to serial attacks are most frequently selected for status. All symptomatic epilepsies dependent upon gross organic brain lesions, such as trauma, abscess, tumor, thrombus, and infantile hemiplegia, are particularly prone to develop status (unilateralis). They usually die in this state ultimately. Status pareticus is an instance of cerebral disease often terminating in status. There is a close analogy between all convulsive phenomena, and the different manifestations of status resulting from different brain lesions must bear a close relationship to the status of epilepsy both from a histocytologic and physiopathologic view-point. Status is a notable ending to all partial Jacksonian epilepsy. A third of the status cases on record have been at bottom dependent upon organic lesions of the cortex. However, its mono- or hemispastic character greatly lessens the possibility of the fatal character of individual status periods. Such cases often have several periods before the fatal one. Local transitory paralysis often occurs in such cases. Jackson and Gowers believe that the latter symptoms are pathognomonic of status; still later data, however, show that exhaustion paralysis is not a constant symptom of status (Clark and Prout).

Sex.—The influence of sex is of little moment in the status production. Lorenz and Bourneville, however, believe women are twice as frequently affected as men, but in one hundred and eleven cases collected from literature, sixty-seven were men and forty-four were women. The mortality rate between the two sexes is about equal (in sixty cases twenty-eight were men, thirty-two were women). If the hereditary factor of epilepsy were of less moment in the pro-

duction of epilepsy proper, it is possible that the difference in sex, including difference of manner of life, occupation, etc., would be greater factors in determining sex preference than at present exists.

Age.—There is no particular age at which status is apt to develop. Its slightly increased frequency at a particular epoch is due to the fact that epilepsy itself develops more especially at certain ages. In rare instances status may develop in earliest childhood. Thus W. Ramsey Smith reports two cases, one of a child of three who died of it, and another of four who had typical status and recovered. Such cases could hardly be ranked with the statistics of idiopathic epilepsy of the adult, and were probably due to gross cerebral disease.

It is the general rule for infantile cerebral palsy to be ushered in by convulsions which are either partial or general with local onset and comprised in serial or status periods; but from a pathogenetic standpoint such cases cannot be regarded as the status of epilepsy *per se*, which develops long after the disease is first inaugurated. While it is by no means uncommon to see status in extremely aged epileptics (of seventy or eighty years), senile patients usually die of status apoplecticus from arterial rupture, sclerotic changes being the primary cause of the epilepsy itself.

Menstruation.—The influence of menstruation is *nil*, much less so even than in inducing epilepsy proper. Usually the occurrence of serial attacks at menstruation in an individual not previously known to be epileptic confirms the diagnosis of *status hystericus* which usually occurs at this epoch. It is, however, most unfortunate that grand hysteria in its status form is at times so intimately blended with the true symptoms of status epilepticus that it is often impossible for even the most expert to make more than a provisional diagnosis until the history and cardinal

curves (fever, pulse, and respiration) are at hand to make certain the diagnosis; happily such problems are of rare occurrence (see clinical charts). A few epileptics have hysteria which in turn develops into status at the menstrual period, while the same patient may have status epilepticus between the menstrual periods. Classic status epilepticus is not more difficult to differentiate from typical status than essential epilepsy from major hysteria, but unfortunately there are
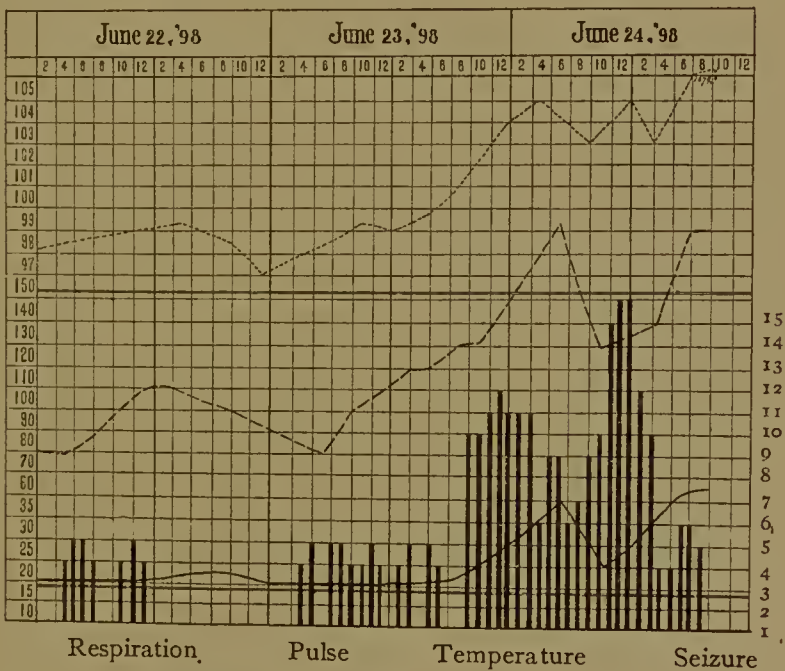
Fig. 9.—Chart showing the clinical relationship between the temperature, pulse, respiration, and epileptic seizures in a typical fatal case of status epilepticus. The hourly record of seizures takes its reading standard at the right.

many variations in the manifestations of both diseases, which the most experienced fail to differentiate (Steffins).

Interval between the First Epileptic Attack and Status Epilepticus.—Of forty-two cases reported from the Craig Colony (Clark and Prout), in nineteen who developed their disease between birth and four years of age, the average interval was eight years; in seven

developing epilepsy between ten and fourteen the average was six; in two developing over thirty the average was twenty-one years. A summary of these
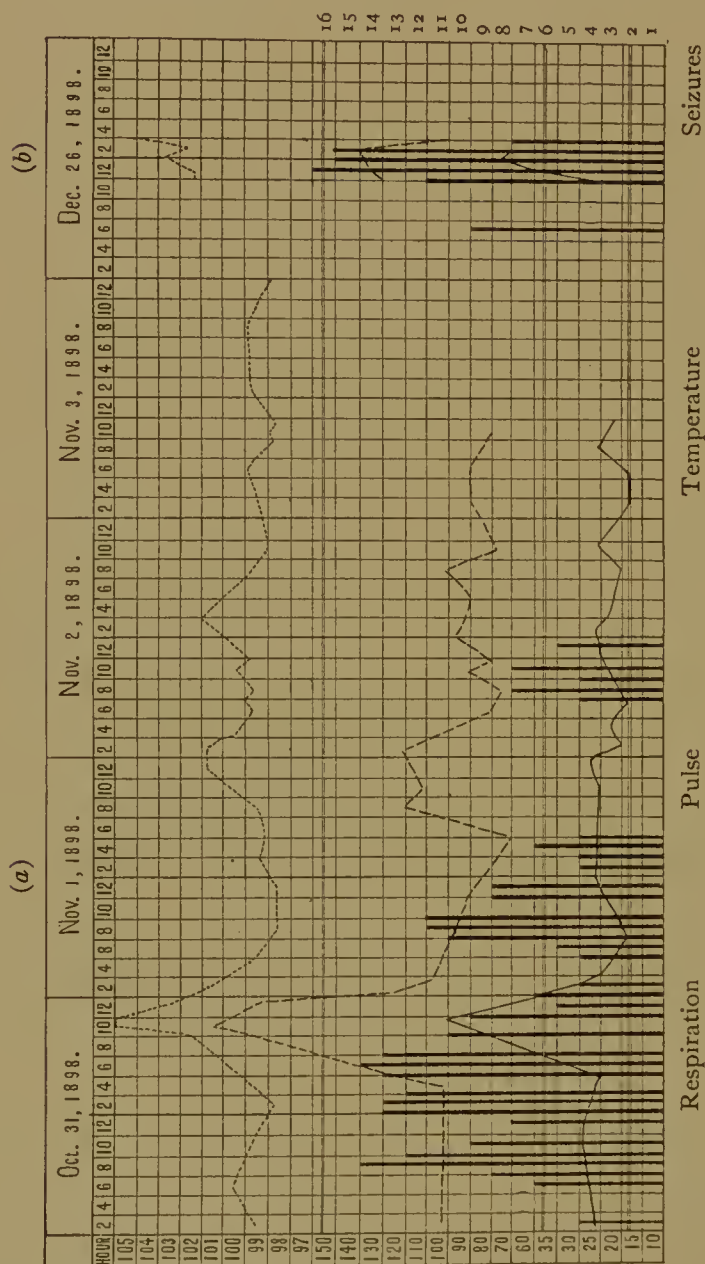
Fig. 10.—Chart showing the clinical relationship between the temperature, pulse, respiration, and epileptic seizures (a) in a typical recoverable case of status epilepticus, (b) in a fatal acute status in the same case, some seven weeks later. The hourly record of seizures takes its reading standard at the right.

facts seems to show that in epilepsies developed in the later epochs of life the onset of status is delayed for a longer period of time than in infancy; the data,

however, are too meager to be more than suggestive. Within general limits it may be said that epilepsy must be well developed before status will supervene. The period of time may vary from a few to many years. In making the prognosis of the occurrence of status in any given cases, predisposition, the excitant, the form of epilepsy, and the course which the disease has already taken, particularly as regards its tendency to exhibit itself in groups or serial periods, must be reckoned with.

Intervals between Status Periods in the Same Case.— No definite time exists between status periods in the same case. A period of days, weeks, months, or years may intervene. The patient may have but one status period and recover not only from the status, but the epilepsy itself, although this termination is rare indeed. Generally one attack of status paves the way for another, and, in fact, there is no limit to the number of status periods that may occur before death supervenes. Although Bourneville and Lorenz state that no individual case may have more than three or four status periods, it is possible for as many as six or seven to occur. In one case the patient had nine typical attacks (Clark and Prout). As a rule, three or four status periods in idiopathic epilepsy cause death, while innumerable periods of status epilepticus unilateralis may occur in hemiplegic epileptics and death not supervene.

In proportion as the individual paroxysms of the status period fall short in point of severity from typical *grand mal*, so does the gravity of that particular status lessen and the more partial or focal (or in which seizures are Jacksonian in the order of beginning), the longer may the individual withstand the exhaustion of the status.

The Immediate Cause of Status Epilepticus.—As status is the climax of all idiopathic epilepsy the im-

portance of this section is lessened. The exciting factors are none other than those subtle causes which underlie the continuity of the disease itself. There are varying degrees of epilepsy, the severity of which are not at all portrayed in the paroxysmal frequency of the disease, for example, as the degree of sensory and mental changes. This fact accounts for the often surprising suddenness with which status supervenes. To be classed with the natural factors in causing status, the artificial one of a too sudden withdrawal or alteration of sedatives should be noted. Status precipitated in this manner is almost incapable of control by a renewed administration of even the most potent sedatives. Probably at least a third of the fatal status cases on record are due to this cause either directly or indirectly.

Inception-symptomatology of the Status.—As a rule, the absence of prodromes is not a striking phenomena, but in the great majority of cases it is heralded by a steady increase of the paroxysmal frequency of the epilepsy either in series or in a gradual daily increase of attacks spread more or less evenly over varying periods of time. A history of serial attacks ranging from five to fifteen or even twenty-five paroxysms in twenty-four hours is the rule. Usually where status occurs in a fulminating manner, the absence of a preceding serial period is explainable on the basis of large doses of bromids, a gradually increased sedation in the face of threatened attacks. Prolonged sedation of bromids alters the natural types of status in many ways.

Clinical Picture of a Typical Status Case.—For the sake of clinical description status is usually divided into two stages, a stuporous and convulsive stage. Usually one seizure of the *grand mal* type follows another of the same character a few minutes or seconds apart. Often the attacks are an hour apart for the

first five or ten seizures. Each attack is complete
and separate, maintaining its peculiar type common
to the individual case. In Jacksonian or, better,
partial epilepsy, the isolated attack holds a distinct
order of invasion so long as exhaustion is not extreme.
At first consciousness is completely regained between
paroxysms; later on, as the periods between shorten,
consciousness is but partly regained, there is a marked
disorientation, and finally the comatose state is not
rallied from between attacks, and the stupor deepens
into profound coma. All cases, idiopathic or partial,
in which an order of muscular march obtains, the subse-
quent coma is less profound and the status in conse-
quence is less severe. As the attacks culminate in their
greatest frequency, the periods of rest between con-
vulsions may be entirely omitted and some part of the
body may remain continually in spasm. The part last
involved in convulsion does not cease from agitation
before the muscles engaged in the initial stage of the
next paroxysm begin again to sweep the rounds of the
muscular invasion of the next fit. The spasm may be
essentially clonic or there may be a slight lessening
of the paroxysmal intensity, thus marking the end
and the beginning of isolated discharges; this over-
lapping is almost always seen in the climax of the con-
vulsive stage of fatal status. With the increasing fre-
quency of attacks the paroxysms usually diminish in in-
tensity, and the tonic period if present at the beginning
may be abbreviated or omitted entirely in the advanced
status. The seizure at the end of the convulsive
stage may be localized to a single muscle or a small
group of muscles. Generally in these status periods
composed of fulminant convulsions (of general and
simultaneous body involvement), the end of the con-
vulsive stage presents only slight general or fibrillary
tremor over the entire body.

As the exhaustion increases after the first few

attacks, there is elevation of temperature, increased pulse rate, and respiratory frequency. The pulse and temperature may surmount to a great height; temperature 107° or 108° F., and pulse 160 to 200 per minute. At last the convulsions lessen in frequency and the stuporous stage is ushered in with coma or collapse, which is quite analogous to the coma of typhus or typhoid. The state is but the resultant exhaustion of the convulsive stage. The mouth is foul, the tongue dry and fissured, and the skin is covered with cold clammy sweat; swallowing becomes difficult or impossible. The urine is usually voided and stools may be passed involuntarily. While the patient may die in the throes of the convulsive stage in fatal status, a few hours are usually passed at least in the stage of coma before death. Even in well-advanced coma slight convulsive tremors may occur from time to time.

All reflexes are abolished in the coma, the respiration becomes loud, noisy, and stertorous in character, and the temperature and pulse may undergo marked alteration depending upon the frequency and intensity of the foregoing symptoms. Death may terminate the stuporous stage at any time. If recovery is to occur, coma wears away and is slowly replaced by stupor which in turn is often followed by mild delirium or hallucinations. The latter state of severe exhaustion is finally replaced by a more or less rapid convalescence and the patient ultimately resumes the pre-status condition in the course of a week. As a rule, if recovery does not follow more or less promptly, a low muttering delirium supervenes; extensive sloughing of the nates follows and life itself is more or less suddenly terminated. The foregoing constitutes in general terms the usual clinical picture of status epilepticus.

Seizures may occur from 50 to 200 a day. The

highest record of *grand mal* attacks for any one case is probably held by one of Leroy's in which there were 488 attacks in twenty-four hours; the patient had 1000 attacks in three days; the status ended fatally. Some American writers have recently alluded to the extraordinary number of attacks which have taken place within a period of several weeks. Thus, Parsons reports 1400 attacks in four weeks, others 2000 and even 3000 in similar periods. Such statistics belong to epilepsy proper, as status does not extend over such a long period of time.

The duration of the convulsive stage is very variable, it may last between three or four hours or it may extend over a period of several days. A case is reported which persisted for one week (Clark and Prout). As a general rule, however, the convulsive stage lasts from eight or ten hours to three or four days. It forms from about a third to a half of the whole status period. The duration of status in its entirety is usually not less than twenty hours or over twelve days, if both stages are present; not infrequently patients may die in the convulsive stage after five or six hours in paroxysms. In 130 cases all were within the limit. If either stage of status is curtailed it is the stuporous. About a third of the fatal cases have no real stage of coma, there being but a few moments' cessation of attacks before the death agony.*

Diagnosis.—When the history of genuine epilepsy is obtainable the diagnosis is, as a rule, comparatively easy by considering the presence or absence of the cardinal symptoms (fever, pulse, and respiratory curves). (See clinical charts for illustrations in diagnosis.)

Prognosis.—The prognosis of status is necessarily grave, knowing it to be the severest form of the epilep-

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* For the pathology of status the reader is referred to the special chapter on this subject.

tic state. A low temperature is not necessarily a favorable symptom, while there have been numerous recoveries with temperatures of 104° and 106° F. Even a temperature of 107° F. is not absolutely prognostic of death as Lorenz holds (Clark and Prout). A patient at the Craig Colony recovered, although the temperature reached 109° F. (Spratling). The individual resistance, the suddenness of onset, and the general severity of the attack must enter into a comprehensive prognosis, as well as a knowledge of the cardinal curves. Extreme paralysis of the pharynx is an unfavorable symptom, but many patients have this symptom well marked and yet recover. Often for a few days after convalescence, the patient still suffers from difficulty in swallowing, which is frequently accompanied by nasal regurgitation.

The duration of the status is not an absolute factor in prognosis, as Bourneville has held; patients may recover after nine, ten, or even twelve days' duration of the disease and die in a subsequent status of a much shorter duration.

Usually status unilateralis is markedly prolonged without being fatal. In proportion as the paroxysm of any status case varies from idiopathic *grand mal* without order of invasion, the possibility of a fatal termination lessens. The shorter the inter-paroxysmal periods of rest between the first attack the worse the prognosis. In such cases acute exhaustion in the convulsive stage is imminent and only the strongest sedatives are of avail. To be able to postpone the paroxysm as soon as the first frequency of the status onset occurs is the strategic point in the treatment, hence the prognosis is based in no small degree upon the efficiency of early emergency treatment. A sudden rise or fall in any one of the cardinal curves, a sudden cessation of convulsions without evidence of returning consciousness are absolutely prognostic of

death. Changes of this sort always occur in fatal status, usually from six to eight hours before death.

Before Lorenz collected the notes of eighty cases from different authors the mortality was held by Nothnagel, Binswanger, and Legrand du Saulle at a little more than 50 per cent. Lorenz placed it, however, at less than 45 per cent. Clark and Prout place it at 33⅓ per cent. in the study of fifty-two cases of status.

It is but just to compute the mortality from status upon the total number of status periods, instead of individual cases treated, which, in the statistics of 100 status periods occurring at Craig Colony, place the mortality at 14 per cent. It may be laid down as a general rule that *the gradual, deliberate, step-like increase of the cardinal symptoms in spite of sedatives renders prognosis as absolutely unfavorable.*

TREATMENT.

Prophylaxis.—The prophylactic treatment of the status is of great importance. If serial attacks are presented in the history, sedatives must be employed to check the periodicity of attacks, which can largely be accomplished by giving bromids at the threatened periods in sufficient quantities to partly suppress the convulsions, thus spreading out or prolonging the period of discharge, and in this manner the exhaustion is lessened for any particular period.

In case there is a gradually increasing paroxysmal frequency, bromids in high dosage must be employed; any sudden decrease or withdrawal of bromids may precipitate status; a large percentage of status cases in general practice are induced by such indiscretions. The long account of suppressed paroxysms is then paid for by a status condition which frequently terminates the patient's life. Cases in which there is a sudden and prolonged suppression of attack need careful

watching, as they constitute a certain percentage of the status statistics. For these cases the emergency prescription for an incipient status should be in readiness. The formula for the emergency prescription has been used with unusually good results. It was first used by me in 1896, and in a slightly modified form it has been constantly employed in all threatened status. The formula and directions for using the same are:

R. Tr. Opii Deod. .... ℳ v.
Potass. Bromid. .... gr. xxv.
Chlor. Hydt........................... gr. xx.
Liq. Morph. Sulph. (U. S.) .... ℥ i.   M.
SIG.—One dose; repeat in two hours if necessary.

An explanation of the efficacy of this prescription might be made on the following principle: The chloral and morphin are the first to act in their respective order, the chloral as a sedative upon the vascular system and especially upon the blood-supply to the brain; the morphin as a sedative on the nerve cells. Following their immediate combined action we obtain the slower and more permanent sedative effects of the bromid and opium upon the cerebral centers.

One experienced in the care of epileptics becomes watchful for the possible occurrence of status, and consequently employs methods in the general treatment of the disease which will lessen the impetuosity of a threatened status period. In chronic epileptics in whom serial or pseudo-status is liable of frequent occurrence, the bromids need to be given between periods with the greatest caution, in order that the full sedation of the salt may be used effectually at the status crisis; in other words, a certain reserve must be held for the paroxysmal climax. The employment of all remedies favorable to the general amelioration of the epileptic state should be the general rule in prophylactic treatment of status.

The surgical treatment of status is relatively unimportant; status of idiopathic *grand mal* cannot be successfully treated by any known surgical procedure, either from clinical or pathologic considerations, and therefore operative interference is not justifiable in idiopathics. Trephining on the basis of the late revival of the old theory—whose ghost has just been laid again—that the status is the result of an increase of intracranial pressure, has not alleviated the convulsions of the status condition to any appreciable extent. However, trephining for the status epileptiformis, caused by a recent trauma, is imperative and its early adoption is attended by the most brilliant results (Mynter and Krauss), but the surgical treatment of this form of epilepsy should never be postponed until the status develops. Operation should be undertaken as soon as the influence of the trauma can be determined. The status of old organic lesions, such as the old infantile cerebral palsies, is hardly operable; it should be considered in the class of idiopathic epilepsy, so far as treatment is concerned.

It may appear from the conservative standpoint of this chapter that status, being but a climax of epilepsy proper, is therefore not preventable until we are able to modify the underlying tissue changes in the cerebral cortex. However, many status periods are aborted by proper treatment of the epilepsy, as well as the status itself. Not only may the life of the patient be saved, but the patient may recover in rare instances from the epilepsy itself. The present mortality from status can be further decreased to a marked extent by resorting to prompt treatment in the convulsive stage. If the seizures cannot be entirely controlled in all cases, it is generally possible to lessen the paroxysmal frequency and thus conserve the organism from acute exhaustion, the most fatal sequence of status. Indeed, the condition of acute exhaustion stands in the

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same sequential relation to death by status as status does to epilepsy proper.

Treatment of the Convulsive Stage.—After the first five or six paroxysms, the emergency prescription heretofore mentioned should be given. In the fifteen or twenty minutes necessary for the remedy to take effect, chloroform should be employed to immediately lessen the severity and number of paroxysms. This anesthetic must be administered to the point of complete surgical anesthesia in order to be effective in controlling the convulsions. *It must always be borne in mind that that which is indicated for the convulsive period is contraindicated for the subsequent stuporous stage; therefore antispasmodics must be given with due caution.*

Delasiauve has recommended general and local blood-letting, drastic cathartics, ice to the head, and quinin by rectum. Bourneville and some other French writers claim to still hold this plan beneficial. Acting upon the theory that venesection lessens toxicity of the abnormal accumulation of waste products (toxins) in the blood, the plan of blood-letting is a good one. Venesection in status finds its greatest value when employed in plethoric epileptics. Status only too frequently occurs in the feeble instead of the robust; usually not more than a third of status cases are in normal bodily vigor at their status periods. A better practice is to venesect and inject saline solution. This method, as it doubly reduces the toxicity of the blood, deserves first place after the emergency treatment of status has been tried and found inefficient. As for venesecting for the supposed increased intracranial and arterial pressure, the condition has no basis in fact; on the contrary, Féré has shown that intracranial and arterial pressure is markedly diminished in status, and Nornatsky and Arndt have recently conclusively demonstrated by the elaborate

experiments upon isolated convulsions, serial and status periods, that the increase of intracranial pressure is only a result and not a cause of epilepsy.

If drastic cathartics are given early, they may be of use; in the later stages they are brought into action with great difficulty. The application of ice to the head in the convulsive stage is to be discouraged, especially so as the coma becomes continuous. It has its advocacy in the early belief that status was a meningitic affection. Ice to the head may be employed in convalescence, as it appears to lessen the post status delirium and often contributes to the comfort of the patient by lessening the headaches and the many morbid cephalalgic sensations. Ice applications to the spine have been highly recommended by Gowers and Crichton-Brown. Bourneville recommends ammonia inhalations for the convulsions; large doses of bromids (from twelve to sixteen grams daily) and camphor. The efficacy of this treatment rests entirely upon the bromid. The administration of camphor and ammonia inhalations are generally attended by negative results. The subcutaneous use of bromids in status is to be highly commended. After extensive trials ranging from a 10 per cent. solution to that of full saturation of the salt, I have determined that subcutaneous injections of bromids should not be given in a stronger solution than 10 per cent.; even then in about a third of all cases abscesses will form; care must therefore be exercised to give the salt under strict antisepsis, and in parts where abscesses can be most easily treated in convalescence. The injections are extremely painful and should be given only when coma is profound and only when the convulsive stage is well advanced. Gentle friction and moist heat at points of bromid injection favors absorption and discourages abscess and necrosis. This plan of giving bromids is also recommended especially by Wilder-

muth, and it has been favorably commented upon by many others. If hypodermic medication of bromid is to be of service, 120 grains should modify the status symptoms in the course of two or three hours. If this amount does not appreciably affect the convulsions, no more salt should be given by this method; sodium bromid is the best salt to administer in this way.

Asafetida, belladonna, bromethyl, and atropin have been used by Bourneville without gaining any good results. Belladonna, although of signal value in epilepsy proper, has too feeble antispasmodic properties to be of any great service in status. Bromethyl is sometimes effective, especially in incipient status. Atropin should be used only in respiratory failure in the stuporous stage. Crichton-Brown introduced amyl nitrate and based its favorable action in the status of epilepsy upon the fact that many of the status symptoms were due to the asphyxia of the brain and that the use of this drug should relax the arterioles and favor the circulatory return. However true the theory may be, I have never seen any benefit by its administration in the severe *grand mal* status, but have observed slight benefits from its use in serial psychic epilepsy, its action here being due to an alteration in the cerebral circulation from increased cardiac activity and dilation of the arterioles. The increased blood-supply in turn excites increased inhibitory control of the sensory cortical cells. On the other hand, cardiac depressants and vasoconstrictors, such as ergot, may have a similar action. Negative and positive states of cerebral dynamics may give rise to an equal amount of cerebral inhibition. These alternating sensory states due to the modification of the circulation are well known to modify the milder forms of epileptic paroxysms. Since Crichton-Brown's advocacy of nitrate of amyl in status, McBride, Berger,

Jolly, and Bourneville have advised against its use in status epilepticus.

Solwith has injected Bonjeon's ergotin in several of his patients with benefit, but Crichton-Brown and a number of others have reported against its use. At best, ergotin is but a feeble spinal depressant (Word), and on rational grounds the status cannot be beneficially modified by the drug. Binswanger has recommended narcotics on rational grounds, while Crichton-Brown regards them as pernicious. Combined with other drugs, such as the bromids, they are of distinct advantage. I have employed them in the convulsive stage of several status cases with marked benefit.

In the past few years amyl hydrate has been successfully used by Wildermuth in six cases. While this drug is uncertain in its therapeutic properties, yet it has no marked depressive action upon circulation or respiration and may be tried in the convulsive stage of status. The following list of drugs not before mentioned in this section have all been tried and each one has its special advocate: Ether, chloroform, chloral hydrate, sulphate of morphin, hydrobromate of hyoscin, salicylate of physostigma, and hydrobromate of cocain. Ether and chloroform are given with a view of immediately checking convulsions by paralyzing motor centers. They are only of temporary value, the better one by far being chloroform. Its inhalation should be undertaken in the early part of status, as it is too depressing upon the heart when there is much exhaustion from a large number of attacks. Ether may be administered in prolonged status, as it is devoid of severe depression on the circulation and respiration. The attacks only disappear in the use of either when given to the point of surgical anesthesia, and the seizures return before consciousness is regained. As heretofore stated, anesthetics should never be employed except when death is imminent in convulsions,

or until slower drugs, such as chloral hydrate and bromids, may be taken in the general circulation. When it is injudicious to employ anesthetics to the surgical point, they may still be used to modify the severity of paroxysms, especially in Jacksonian seizures, or in status unilateralis following organic brain disease.

The use of chloral in status has many advocates and deservedly so, but when it is given in advanced status in from sixty- to ninety-grain doses uncombined with other drugs, it is of doubtful value. Large doses of chloral uncombined with cardiac stimulants should always be given with extreme caution in advanced status. Forty grains of potassium bromid and thirty grains of chloral by rectum, to be repeated in three hours, if convulsion continues, is one of the routine treatments which I have employed. Chloral must be given early and before cardiac failure is imminent. At the first indication that the sedatives are becoming effective in controlling the convulsions, the dose of the drugs should be modified accordingly. Hypodermics of a quarter of a grain of morphin combined with the foregoing sedatives is often desirable, but the action of morphin given alone is too uncertain to be of signal value. If the convulsions produce great asphyxia and cyanosis, inhalations of oxygen may be given with advantage; it aids the circulation, respiration, and urinary secretion.

A 10 per cent. solution of bromin in an emulsion with the oil of sesame is an excellent sedative for the status, given by rectum or hypodermically, but to be effective in severe status it must be given in large amounts. Its sedative action is about half of that of the bromid salt, volume for volume. The after-toxicity from the bromin sedation is much less than from the bromids, but bromin is much slower in action than the bromid salts and, as I have already pointed

out, the early sedation in the convulsive stage is of paramount importance.

Chloretone has been used in status to some extent in place of chloral, as the latter is very dangerous on weak hearts. However, chloral is largely efficacious in status because of its marked sedation on the cerebral circulation as well as on the brain itself. Chloretone not having this sedative power over the circulation, is much less valuable than chloral; besides it produces on its own account, in comparatively small doses, a toxicity and a resultant delirium most pernicious. Chloretone cannot supplant chloral in the treatment of the status epilepticus.

Innumerable other remedies for a time have had their place in status treatment, but they never have seriously endangered the high regard in which chloral and bromid have been held. Status epilepticus is a state above all others in which weak and ineffectual compounds must be cast aside, as the time element in gaining control of the patient's disease is of paramount importance. Chloral ranks first just as pre-eminently in the treatment of the convulsive stage of status as bromid does in the medication of epilepsy proper.

To summarize our plan for the treatment of the convulsive stage: The "emergency prescription" should be administered after the first six attacks, either by mouth or by rectum; later, if status continues, use chloroform and continue bromid and chloral by rectum, or hypodermics of bromid. *Only so much sedation must be employed as may be necessary to control the severity and number of convulsions.*

The treatment of the stuporous stage of status largely depends upon the severity and proper treatment of the convulsive stage. The treatment must be supportive. The heart and lungs need close attention, not only for the acute exhaustion entailed by the previous convulsive period, but for complications that

are apt to occur in the lungs.   Alcohol should be used
freely during the exhaustive state of coma.   Although
the cold bath will favorably alter the fever curve for
a time, its action is rarely of lasting value.   Undoubt-
edly the prolonged action of the sedatives given in the
convulsive state is in no small degree responsible for
the severity of the coma; therefore, as a rule, drugs
counteracting the antispasmodics should be given in
the stuporous stage.   The effects of chloral are quite
easily overcome by whisky, digitalis, and strychnin,
but the slower and more permanent sedation of bromid
is not so easily counteracted.   As soon as the ex-
haustion of intestinal peristalsis is recovered from
sufficiently, diuretics and cathartics may be given.
The arterial pressure which is always much lowered
in the stuporous stage should always be increased by
hypodermoclysis or enteroclysis, as a routine plan of
treatment.   When Cheyne-Stokes respiration is ex-
treme, faradization of the phrenic nerve as advised
by Wildermuth may be tried.

To summarize the treatment of the comatose period:
Counteract the exhaustion and sedation of the con-
vulsive stage and watch for a possible return of con-
vulsions; in the later stages, stimulate and support the
patient and treat complications promptly.

The treatment of the post-status period is generally
supportive.   Usually there is a freedom from attacks
for a more or less longer period of time, but this is too
uncertain to warrant an entire withdrawal of sedation.
Occasionally the convulsion may return in the post-
status period and cause death, as the weakened con-
dition of the organism is unable to withstand the
renewed onset.   For the delirium and the milder type
of mania in the post-status stage $\frac{1}{20}$ of a grain of
hyoscin combined with $\frac{1}{6}$ of a grain of morphin may
be given.   It should be given often enough to keep
the patient quiet and free from the motor restlessness

which so frequently prolongs and delays convalescence. Insomnia is commonly a troublesome symptom in the post-status period; it is best controlled by small doses of morphin combined with paraldehyd, trional, or chloretone. Hot baths and hot milk at night are often sufficient to overcome mild states of insomnia. Too little stress has been placed upon the milder remedies in restoring normal sleep, and especially is this true in the insomnia after status; its rule of treatment should be largely comprised in rest and food. The post-status mania rarely necessitates treatment by physical restraint. But such agents, if necessary, are best employed at the patient's home; mental aberration is almost always transient. Epileptics who may be insane in inter-status periods need to be watched carefully after status, as they are frequently suicidal and occasionally homicidal. If the patients do not exhibit great violence at the inception of the convulsive stage, no special change in the surroundings of the patient need be made; but if the contrary obtains, the patient's mattress should be placed on the floor in such a manner that other mattresses may be used for side pads to break the furious onset of convulsions. No epileptic should be physically restrained in the convulsive state, as it only excites and exhausts. A roll of cloth should be employed to prevent the incessant biting of the lips and tongue.

The general care and nursing of a status case is fully as important as the medicinal. The temperature and pulse should be recorded hourly to gauge the degree of individual exhaustion occasioned by isolated attacks. The nurse needs to make note of the convulsive phenomena; order of muscular invasion, if any; length of time of tonic and clonic spasm; the presence and absence of the typical symptoms of *grand mal*, etc., as the treatment is always based upon the bedside data. The kind and intensity of convulsive phenom-

ena are only to be actually determined by grasping
the muscle involved in different stages of an attack;
the eye is always deceptive, and many reported atyp-
ical phenomena of the status have their basis in the
latter form of faulty observation.

The administration of proper and sufficient food in a
status case has been found to materially aid in saving
the patient's life.   Foods should be in liquid form and
highly nutritious from the start.   Various preparations
of milk, eggs, and beef extracts may be given; but
plain peptonized milk is by far the best food of all.
It should be given often and in small amounts.   All
foods should be given early, before exhaustion and
coma are profound.   The earlier the food is given, the
greater the chances are that its absorption will be
consummated.   As heretofore stated, pharyngeal and
laryngeal paralysis is present in some degree even
before coma is continuous between paroxysms, there-
fore many terminal bronchites, lobular and lobar pneu-
monias are avoided by judicious feeding.   Every
attention should be directed toward preventing the
inhalation of food or vomited matter from forced
feeding.   As the difficulty of swallowing becomes pro-
gressively more marked, forced feeding by a nasal or
mouth tube may be resorted to.   Occasional lavage of
the stomach may be practised before the feedings.
However, on the whole, the great distress to the patient
and the liability to interruption by seizures render
the routine practice of forced feeding of doubtful value;
I cannot recommend it.   As a last resort, nutrient
enemata may be given by rectum. The patient absorbs
an infinitesimal amount of nourishment taken by the
rectum in status, as secretive and absorptive activity
are at a minimum.   Postmortem in status cases usu-
ally discloses a quantity of undigested food through-
out the intestinal tract.   The little possibility that
remains for rectal absorption should be taken advan-

tage of for rectal medication and enteroclysis. The patient should be given plenty of water, a fact frequently overlooked in the confusion of treatment; often the first conscious demand of the patient is for water.

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CHAPTER VIII.

FORMS OF EPILEPTIC AURA.*

Their Meaning and Significance. Their Classification. Sensory. Psychic. Motor. Irregular. Special Sense Aura. Illustrations of Each. The More Common Forms and Types.

Meaning and Significance.—In its literal meaning the word aura signifies "vapor or emanation from a body, surrounding it like an atmosphere"; though in the sense in which it is used to denote a warning or a symptom of an approaching attack, it has come through usage to have a much broader clinical application. It was first used by Pelops, the master of Galen, who thought that the fit began in the form of a "spirituous vapor" in the veins of the extremities and ascended to the head, whereupon the patient became unconscious.

Much obscurity still surrounds the nature and value of the aura epileptica; and while its interest is great, its practical value is at present unfortunately too often limited to aid in the detection of the approach of attacks which might otherwise be unperceived until the convulsion was actually in progress. Future enlightenment may give them an etiologic value they are not now known to possess.

For a long time it was held that the nervous impulse or irritation causing the spasm began in the part where the aura first occurred, though now it is generally held that in the vast majority of cases the initial impulse begins in the central nervous system, the

* From an etymologic point it would be more correct to use the classical plural, either the Latin *Auræ* or the Greek *Aurai;* but aura has secured so firm a footing in medical English that it seems useless to employ any other term.

sensation being referred only to the periphery.   At the same time, there may be an occasional case in which the impulse starts at the periphery, as has been shown by the cessation of the attacks when the local irritation, such as adherent prepuce or a stricture of the urethra, was removed; or, again, when the tying of a cord tightly about the member affected checks the progress of the fit.   But in all such cases the question arises whether we ought to designate the attacks as epileptic, or simply as reflex, or epileptiform.

It is well known that disease of the brain may cause descending degeneration in the spinal cord, and ascending disease in the spinal cord may produce disease of the brain.   Experiments have proved that any injury in the periphery may not only cause changes in the central nervous system, but structural alterations in nervous tissue and functions in other parts of the periphery as well.

Brown-Séquard showed this to be true when he cut the sciatic nerve in the leg of a guinea-pig and caused structural alteration in the skin of the face, afterward producing epileptiform convulsions by irritating certain portions of the face, then removing the basis of the attacks by excising this epileptogenous area.

We may also mention in this connection the referred sensations in coxalgia in which the pain is sharply felt in the knee and ankle-joint, and the acute gastric pains that follow vertebral disease.

There has been a growing tendency for some years to regard the aura as constituting an essential part of the epileptic attack.   This seems entirely reasonable, fully as much so as it is to regard fever and congestion as preliminary parts of pneumonia, when pneumonia follows.   Sometimes the aura arises and persists for an indefinite period and disappears without an attack following; just as there may be fever and congestion of the lungs without pneumonia following.   It is,

therefore, in our opinion, a good plan to look upon the aura as the beginning of the fit, not simply as a warning of its approach, and to treat the case accordingly.

Unfortunately, this cannot often be done, for it is rare for the physician to see the patient before the convulsion has occurred, the aura being not far removed from the attack itself, generally not more than a few seconds, occasionally half an hour or so, while in very exceptional instances the period is longer.

Classification.—We can best study the various aura by grouping them under four heads, as follows:

Sensory.
Psychic.
Motor.
Irregular.

The first includes some disturbance in the patient's sensation; the second, some disturbance in mentality; the third, some disturbance in the motor system; while the fourth embraces elements of two or more, or even of all of these.

It is difficult to say which of these forms of aura possess the greater clinical importance, though, because they so greatly outnumber all the rest, we incline to attach the greatest weight to those of a sensory nature; those in which a feeling of some kind, a numbness, or tingling, or pain, or a feeling of discomfort, is felt for variable periods before the fit comes on.

In a close analysis of 815 men and 510 women for the establishment of individual aura, it was found that 319 men, equal to 36⅓ per cent. of the entire number, and 186 women, equal to 36½ per cent. of the entire number, had a sensory aura of some kind. It is worth noting how remarkably even this form of aura ran parallel in the two sexes.

In the same cases, 33 men and 19 women had psychic aura, the 52 cases in both sexes constituting less than

4 per cent. of the entire number, while 22 men and 10 women, 32 in all—a fraction over 2 per cent. of the total—had a motor aura of some kind. Combining all who had an aura of some kind, we have a total of 589 out of 1325, equal to about 45 per cent., leaving 55 per cent. who had no aura.

It appears from this that in less than half of all cases there is a specialized aura, though it is best not to regard this as conclusive, for it is reasonable to suppose that some epileptics of low intelligence experience a sensory aura which they are unable to describe. Allowing for these, it is safe to say that 50 epileptics out of every 100 have an aura of some kind.

This proportion is much greater than that mentioned by Echeverria, who states that a perceived aura was found in 10 per cent. of the men and $11\frac{1}{2}$ per cent. of the women in the 306 cases studied by him.

Gowers classifies the aura under seven groups, in substance as follows: " (a) Unilateral; (b) certain general aura, such as sensations in the limbs, tremors, faintness, etc.; (c) aura referred to certain organs, especially those supplied by the pneumogastric nerve, most of the visceral warning coming under this head; (d) vertigo and allied sensations; (e) certain sensations in the head, pain, etc.; (f) psychic aura, the consciousness of an emotion or idea; (g) special sense warnings, some of these being unilateral." In 1000 cases he found—

1. A unilateral aura in 86, topographically distributed as follows: in the arm in 45 cases; in the leg in 15; in the face in 17; in the tongue in 7; in the rump in 2.

2. Bilateral general aura in 74 cases: in the arms in 11; in the legs in 12; shivering, trembling, nausea, and weakness in 51.

3. Aura affecting organs in 106 cases.

4. Fainting and dizzy aura in 90 cases.

5. Head aura in 50 cases.

6. Psychic aura in 25 cases.

7. Aura of the special senses in 119 cases.

A total of 600 epileptics out of 1000 cases observed by Gowers had an aura of some kind.

According to Binswanger, Herpin found an aura of the sense organs in 27 per cent. of all cases; while Bennett found an aura of some kind in 34.4 per cent., the latter finding the aura relatively rare in major epilepsy and far more frequent in the milder forms,—a fact verified by my own experience.

Voisin * and Féré † classify the aura under four heads: Motor, sensory, sensorial, and intellectual, following that used by Delasiauve, but neither gives an estimate of the proportion of each. Féré states: "It (the aura) is very frequent; O'Connor has found it in 78 cases out of 100."

According to Féré, Thorion claims that the epileptic crisis is preceded by a greatly increased elimination of the urinary elements, a veritable discharge; this chemical aura, he thinks, might even enable one to predict the return of the attack.

Sensory aura are vastly more common than all the rest and partake of the greatest imaginable range in character. They may be confined to the special senses. and include variable degrees of perversions of taste, smell, sight, touch, and sound. Any abnormality in the functions of the special sense organs in the nature of an aura may be fleeting and transitory, or may persist for hours before the fit comes on.

Visual aura greatly predominate, occurring as often as those of taste, hearing, and smell combined. They usually take the form of flashes of light, the colors of the rainbow passing in rapid succession across the

* "L'épilepsie," 1897, p. 54.

† "Epilepsy," "Twentieth Century Practice of Medicine," Vol. x, p. 593.

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field of vision.   In other instances they appear in the nature of optical illusions, people, dogs, cats, and wild animals of various kinds being engrafted on the visual field; while it still more rarely happens that temporary blindness immediately precedes the attack (*amaurosis epileptica*).   In some instances the sudden loss of sight may alone constitute the entire attack, the area of the discharging lesion being so circumscribed as to produce no other results.   Many cases of persons suddenly stricken blind doubtless are due to this cause.

The following instances taken from the author's cases are typical of the more common forms of visual aura:

"Flashes of light, then blackness."

"An object passes before the eyes from five to ten minutes before the attack."

"Dark objects move before the eyes."

"Blurred vision."

"Flashes of blue and red."

"Flashes of light of the rainbow colors."

"At times sees colors."

"Sees wavelike motions in the air just before the fit."

"At times have loss of vision."

"Visual percepts of light."

"Vapor before eyes and numbness in paralyzed hand."

"Eyes get dim."

"Visual aura at times—sees a policeman."

"Partial blindness."

"Sees black, then a yellow light."

"Occasionally a visual aura—sees stars; at other times the aura is epigastric."

"Sees some animal approaching."

"Everything looks bigger than it ought to be just before the fit; people, chairs, and tables are magnified."

According to Sir William R. Gowers, it is not sur-

prising that when central instability exists the visual
center should often lead the way in the sudden de-
rangement of balance, for light constitutes the most
rapid form of motion of which we have any perception,
consequently it must be associated with a correspond-
ing degree of delicacy in the central stimulus that
receives new impulses produced by waves of light.

In addition to the forms of visual aura in which
colors predominate, other sensations and conditions
occasionally affect the eye just before a fit, one of
these being diplopia, or double vision. A case of this
kind was mentioned by Gowers, in which the diplopia
was associated with an aching in one eye and an ap-
parent magnification of objects, things appearing
twice their usual size.

The curious condition of objects seeming to in-
crease or diminish in size is seldom encountered. This
phenomenon when present is explained by either an
increase or diminution in the sensitiveness of the
visual center, which produces an effect on conscious-
ness analogous to that produced by the stimulation
of a larger or smaller area of the retina by a larger
or smaller image.

A male epileptic, twenty-four years of age, who
came under my care, had this aura, objects about him
increasing greatly in size from ten to twelve seconds
before the fit appeared.

In a few cases there is a sensation of pain in the eye-
ball, due probably to some irritation of the fifth nerve,
these cases generally having a motor element in them,
such as twitchings of the muscles about the eye.

Gowers * reports a case in which autopsy was per-
formed, and in which the fits were preceded by a flash
of light or pain in the eye. The autopsy revealed the
presence of a tumor in the occipital lobe, which ex-
tended as far forward as the angular convolution.

* "Epilepsy and Other Convulsive Diseases," p. 79.

In addition to all these, there have been reported cases of highly specialized visual sensations in the form of complex visual conception, or psycho-visual aura; that is, a psychic aura crowding itself in upon the patient's field of consciousness coincident with the appearance of some distorted visual percepts. For instance, a woman always saw bright lights—red, green, and yellow at first; the lights continued, and she saw a girl, "and tried to get to her to ask what the lights meant," and then fell in a fit.

When one color only is seen, it is most apt to be red or blue; when a number follow, the most prominent order of appearance of the colors is red, blue, green, yellow, and occasionally purple. This order is not in harmony with the physical relation of colors, nor does it correspond to the areas of retinal perception, for red, which is most often seen, has one of the smallest retinal fields. On the other hand, it does correspond to a certain extent with the degree of visibility of colors, for Gowers credits Cohn with having observed that the order in which spots of color one millimeter in size can be seen in direct sunlight at a certain distance is as follows: red, blue, green, yellow, and violet, which seems to be very nearly the order in which they appear as aura in epilepsy.

Occasionally we find auditory and visual auræ associated, though this combination is rare, while we have knowledge of one case in which there was complete unilateral loss of hearing just before the fit.

Auditory aura partake, as a rule, of the nature of the following hallucinations:

" Roaring and voices heard two or three minutes before the convulsion appears."

" Roaring in the ears."

" Sound of sea waves in the ear before mild attacks."

" Roaring and buzzing in the ears."

"Noises in the head for a brief period before the attack."

"Roaring followed by a sensation of falling."

Auditory aura occur about a fourth as frequently as the visual and are generally present in from 2 per cent. to 3 per cent. of all cases.

In addition to the forms of auditory disturbance enumerated above, the patient may hear sudden sounds of almost any nature, like the crash of a heavy falling body, the blowing of a whistle, the ringing of a bell; while more rarely there may be the sounds of voices or of music alone.

L. Pierce Clark* quotes Santé de Sanctis and A. Christiani as reporting three cases in which the patients indulged in a variety of songs while undergoing mild convulsive movements; two of them kept time to imaginary music with rhythmic movements of the body, each showing some clonic muscular movements in the lower extremities, while the third sang a single tune, accompanying it with movements imitating an orchestral direction.

The writer is familiar with the cases of two young adult male epileptics, one of them the victim of an infantile palsy affecting the right hand and foot, both of them subject to *grand mal* as well as *petit mal* attacks, the latter form not infrequently being announced by a period of involuntary whistling. I had occasion to witness this phenomenon in one of them five times in one day. The first indication of an attack was a sudden cessation of all movement on the part of the patient; he would stop and stand still if walking, assuming a confused, puzzled expression, when he would grunt quite audibly two or three times, and then whistle a bar from a popular song of the day, repeating it with the next convulsion in the most precise manner. He was fully cognizant afterward

* "Medical News," Sept. 1, 1900.

of what he had done, as shown by the fact that he always turned to me with the question, "Did you notice that one?"

On one occasion he caused a bank cashier, with whom he was dealing at the moment, much astonishment and concern by suddenly repeating the usual grunt two or three times, then whistling a bar from a popular opera while arranging an important financial transaction. None of these anomalous states lasted more than from five to seven seconds. At times they appeared as epileptic equivalents, at others as forerunners of more pronounced convulsions.

I recall two cases in which mild seizures were accompanied with complete auditory deafness, vision remaining normal, so that signs made to the patient to do certain things were immediately obeyed, while spoken commands were unnoticed.

Smell and Taste.—Extraordinary sensations in connection with smell and taste are generally met with, which take the greatest possible range in character and are generally of an unpleasant nature.

Gowers thinks it significant that flavors which are perceived only through the olfactory nerve seem to be associated with taste in the central discharge which causes the warning, the flavor in this instance being of a kind that enters the posterior nares, causing a simultaneous stimulation of the true gustatory nerve.

Some epileptics experience a bitter taste, like quinin, while others have a metallic taste in the mouth that sometimes persists for an hour or more before the convulsion. One patient complained of a feeling of "numbness in the tongue," as though that organ had been completely cocainized. Watson mentions an epileptic who could abort an approaching attack through voluntarily biting his tongue quite severely, doubtless having first experienced an unusual sensation of some kind in it.

Hughlings-Jackson * has drawn attention to a group of cases in which there is at the onset of the paroxysm a crude sensation of smell or of taste, accompanied by movements of chewing, smacking of the lips, and sometimes spitting, all being ascribed to disturbances in the cortex. Some of these are foreshadowed by an epigastric warning.

A. T. states that he almost always has light attacks following severe ones at night, and following the light attacks he has a peculiar taste and sensation in the mouth which he describes as follows: The first is a "sour taste"; the second, similar to that of "wheat bran in the mouth"; the third, a feeling of "stringiness," combined with "numbness in the upper part of the mouth"; the fourth, a "sickish sweet taste" sufficient to produce extreme nausea; the fifth, a "filthy nasty taste" which is extremely disagreeable; the sixth and last being a feeling of "sliminess" in the mouth. The morning following the patient feels "unusually well," much more so than during any time more distant from his seizures. These disorders of taste may continue for a day and gradually shade out from the sixth condition to that of normal sensation. They have persisted for years, and cannot be ascribed to stomachic indigestion.

According to Ferrier, the senses of taste and smell are located in the *uncinate gyrus*, since reflex irritations of this gyrus in the animals he experimented on produced "reflex" movements analogous to those just mentioned as occurring in the human subject, prior to or as a concomitant of lesser epileptic convulsions.

In the case of a male epileptic of forty-seven years the convulsions, following alcoholism, nearly always had an aura which he described as "a snuff of bad air"; the fit appeared after two or three inspirations that were charged with this disagreeable odor.

* "Lancet," Jan. 14, 1899.

The epigastric aura is more commonly met with than all the other sensory aura combined, its presence having been noted in some form in 200 cases, equal to 15 per cent. of the 1325 studied.

The nature of this aura is fairly constant, for it quite regularly appears in one of three forms: The first and most common is that of "a gnawing, indescribable, indefinable sensation at the pit of the stomach," which lasts anywhere from a few seconds up to, but rarely beyond, half an hour before the fit is established; the second is "a deep-seated burning pain"; the third, "a feeling of nausea which grows in intensity and rises like a choking sensation toward the head, until the sensation reaches the throat, when consciousness is lost"; the latter closely resembles the *globus hystericus*.

The exact location and cause of the pain and uneasiness in these cases is entirely problematic, but most likely they are due to some disturbance involving the centric origin of the vagus nerve. This nerve has a wider distribution than any other of the cranial nerves, supplying, as we know, the organs of voice and respiration with both motor and sensory fibers. Its immediate communications are also extensive and remote, while at the seat of its origin it has control over the two most vital processes of life—respiration and circulation.

The rhythmic sequence of the respiratory movements is apparently due to periodic discharges from the respiratory center located in the floor of the fourth ventricle, on each side of the median line, the two halves being intimately connected by commissural fibers. The cause of this periodicity is obscure, but since it has been shown * that the rhythm continues after the combined section of the vagi and glosso-pharyngeal nerves, the spinal cord in the lower cervical

* "American Text-Book of Physiology," p. 458.

region, the posterior roots of the cervical spinal nerves, and the separation of the spinal bulb from the parts above, it indicates that it is inherent in the nerve cells and is not caused by external stimuli carried to the center through afferent nerve fibers.

Loewy has shown that under the above circumstances the rhythm is due to the blood "which, while acting as a continuous excitant, causes discontinuous or periodic discharges," and since the blood-supply is under nervous control, the respiratory center is automatic only with reference to external nerve stimulation.

The problem in epilepsy that rests on this point is to determine whether the initial disturbance, *the influence that breaks up the rhythmic periodicity of the respiratory impulse*, is to be sought in the respiratory center primarily, or whether it lies somewhere in the periphery, in the stomach, in the heart, or in the circulation, and is carried by some vital process to the seat of respiratory life.

It is unusual for the epigastric aura to be referred to any other locality than the immediate region of the stomach. In one case the pain first appeared in the right iliac region, and was always attended with a feeling of nausea. It is noteworthy that, while the vague sensation which nearly always accompanies the epigastric aura passes upward to the throat and head, the pain remains stationary in the region of the stomach. As a rule, consciousness is lost as soon as the aura reaches the head.

Many patients who exhibit an epigastric aura suffer from indigestion which makes its appearance, to some extent, periodically; other stomach disorders are—distention, gastric catarrh, and flatulence.

It seems now a problem as to whether these conditions precede or follow the initial manifestations of the fit, and on this point I differ with Sir William R. Gowers, who says: "We are not justified in assuming

that flatulence is the cause of the attack any more than we are in regarding gastric disturbance as the cause of an attack of migraine which subsides with vomiting." It seems rational to hold that, while the flatulence itself may not include the attack, the conditions which caused the flatulence may be to blame for it, for the reason that correction of disturbances in the processes of nutrition often lessen or entirely remove the cause of the attacks.

All this points to the importance of studying chemic pathology as a cause of epilepsy—a vast, prolific field as yet but little explored, and still less understood.

THE EPIGASTRIC AURA IN FIFTY OUT OF TWO HUNDRED CASES, PRESENTED TO SHOW THE CHIEF FEATURES OF THIS FORM OF WARNING.

No.	Age at Onset of Epilepsy.	Character of Attacks.	Nature of Aura.	Time between Aura and Attack.	Frequency of Attacks.
1	28 years.	G.M.-P.M.	Pain in the upper part of the abdomen.	5 minutes.	About 2 per month.
2	9 "	G.M.	Feeling of faintness in the stomach.	5 "	12 per month.
3	36 "	G.M.	Distressed feeling in the stomach.	5 "	10 " "
4	1½ "	G.M.-P.M.	Faintness in the stomach, rising in the throat, and then roaring in the ears.	2 "	12 " "
5	24 "	G.M.	Something rising from the stomach to the throat, like a ball or lump, producing a choking sensation.	½ minute.	9 " "
6	18 "	G.M.-P.M.	Pain in the stomach, spreading over the body.	5 minutes.	3 " "
7	17 "	G.M.	Pain in the stomach.	10 "	8 to 12 per month.
8	35 "	G.M.	Pain in the stomach, spreading to the lower part of the abdomen.	1 hour.	16 to 20 " "
9	11 "	G.M.	Burning sensation in the stomach.	1 day.	2 to 3 " "
10	13 "	G.M.	Sensation of something rising from the stomach to the throat and head.	2 minutes.	7 per month.
11	4 "	G.M.	Burning sensation rising from the stomach.	1 to 5 minutes.	3 " "
12	20 "	G.M.	Sensation of pressure on the stomach, passing to the throat, producing a choking sensation.	1 to 2 minutes.	4 " "

No.	Age at On-set of Epilepsy.	Character of Attacks.	Nature of Aura.	Time between Aura and Attack.	Frequency of Attacks.
13	21 years.	G.M.-P.M.	Sickness at the stomach; then a fluttering feeling which goes up to the throat, causing a choking sensation.	1 minute.	8 per month.
14	3 "	G.M.	Burning pain in the stomach, rising to the throat, then to the head.	1 "	1 " "
15	16 "	G.M.	Pain in the stomach.	1 hour.	4 " "
16	13 "	G.M.	Sensation of something rising from the stomach to the throat.	15 seconds.	10 " "
17	25 "	G.M.-P.M.	Sensation of gas rising from the stomach, causing oppression and a smothering feeling.	½ minute.	10 " "
18	14 "	G.M.	Stomach feels full of gas. Oppression about the heart.	1 minute.	4 " "
19	7 "	G.M.-P.M.	Nausea. Something rises to the throat. Wants to drink.	1 "	8 " "
20	20 "	G.M.-P.M.	Sensation of a ball rising from the stomach to the throat, producing a choking sensation.	1 to 2 minutes.	12 " "
21	12 "	G.M.	Nausea and distress in the stomach.	2 to 3 hours.	5 to 10 per year.
22	9 "	G.M.	Burning sensations in the stomach, passing up to the throat, producing a choking sensation.	2 to 5 months.	20 to 30 per month.
23	12 "	G.M.-P.M.	Faintness; then a sensation as if something was crawling from the stomach to the throat, with a choking sensation.	1 to 2 "	12 " "
24	11 "	G.M.	Sensation of something rising from the stomach to the throat.	½ minute.	9 " "
25	3 "	G.M.	Indescribable feeling beginning in the abdomen and spreading upward.	5 to 10 seconds.	7 to 10 " "
26	2 "	G.M.	Faintness in the stomach.	5 to 10 "	7 to 10 " "
27	4 "	G.M.-P.M.	Sensation of fullness in the stomach passing up to the throat and mouth.	5 to 30 "	10 " "
28	4 "	G.M.	Burning feeling in the stomach, passing to the heart and throat, followed by a choking sensation.	1 minute.	1 " "

No.	Age at On-set of Epilepsy.	Character of Attacks.	Nature of Aura.	Time between Aura and Attack.	Frequency of Attacks.
29	2 years.	G.M.	Pain in the epigas-trium, rising to the throat and then to the head. Sensation of a ball in the throat.	2 minutes.	3 to 10 per month.
30	23 "	G.M.-P.M.	Pain in the stom-ach and a flutter-ing feeling in the abdomen.	5 "	3 to 10 " "
31	3 "	G.M.	Sensation rising from the stomach to the throat.	½ minute.	10 " "
32	5 "	G.M.-P.M.	Nausea and "bad feeling" in the stomach.	1 minute.	9 " "
33	6 weeks.	G.M.	Feels something move from her right side to the stomach; then rises to the throat and head. Pain in the stomach.	1 to 5 minutes.	6 " "
34	7 years.	G.M.	Tremor in the stomach.	1 to 3 hours.	12 " "
35	3 "	G.M.-P.M.	Pain in the abdo-men, rising to the head.	1 minute.	7 " "
36	33 "	G.M.-P.M.	Sensation of some-thing like a ball rising from the stomach to the throat.	2 minutes.	7 to 10 " "
37	3 "	G.M.	Pain in the stom-ach.	2 to 3 minutes.	30 " "
38	11 "	G.M.	Sharp pains in the stomach.	1 hour.	1 to 2 " "
39	7 months.	G.M.	Pain in the stom-ach.	1 to 2 hours.	1 to 2 " "
40	14 years.	G.M.	Sensation of some-thing rising from the stomach.	1 to 3 minutes.	1 to 5 " "
41	13 "	G.M.	Bad feeling in the stomach, rising to the throat.	1 minute.	4 " "
42	24 "	G.M.	Burning sensation rising from the stomach to the head, producing a choking sensa-tion.	½ to 1 minute.	7 " "
43	12 "	G.M.-P.M.	Nausea and ver-tigo.	1 hour.	1 to 5 " "
44	Infancy.	G.M.	Sick at the stom-ach, and dizzi-ness.	1 "	12 to 16 " "
45	33 years.	G.M.-P.M.	Sensation of some-thing rising from the stomach to the throat.	½ minute.	10 " "
46	13 "	G.M.	Pain in the stom-ach.	2 minutes.	12 " "
47	5 "	G.M.-P.M.	Nausea; then something rising from the throat to the head.	1 to 3 minutes.	22 " "
48	18 months.	G.M.	Pain in the stom-ach and vertigo. Sensation of some-thing rising to the throat.	1 minute.	2 to 8 " "
49	11 years.	G.M.	Pain and fullness in the stomach.	1 "	4 " "
50	6 "	G.M.	Nausea and ver-tigo.	1 hour.	1 " "

Psychic aura may appear in the form of a sudden abnormal mental state, leaving a definite impression on the memory, due to the fact that brain processes which attend mental states may leave a residual effect which makes it possible for their subsequent revival, the perception of which we are apt to refer to as "psychic sensations."

The most common form assumed by the psychic aura is the sudden acceleration of the imagination; a quick overflowing in the processes of thought, in which the train of the imagination, ever rapid, is rushed ahead with trembling, excited haste, until the thread is snapped and unconsciousness occurs.

In some cases this stimulation of the imagination may be preceded by confused ideation, with different memory pictures appearing momentarily in succession.

Romberg mentions a young man with epilepsy, who for nine years had the same recurring thought which was never quite clear to him and which caused him great unrest. He endeavored to free himself from the idea which took the same course every time, and disappeared with the beginning of the attack.

Binswanger states that "similar observations in which, in each attack, definite memory pictures appeared in 'glaring psychic illuminations' are still found in the literature."

When such sensations precede an epileptic fit, they may also be in the single form of an emotion or an idea, the former generally being in the form of fear, an apprehension of something evil about to occur, the patient being genuinely frightened and attempting to run away. In some of the illustrative seizure instances noted in the last chapter, it was stated that the patient's face wore "a terrified aspect," and that in one or two instances the patient tried to run away from impending danger that seemed to him to be present. The presence of this feeling or danger is also

testified to by patients who involuntarily show fight when interfered with while in a seizure, or just as they are going into one.

I have in mind numerous instances of this kind, one of them being a young man subject to frequent attacks of psychic epilepsy, during some of which he would appear pale, confused, and ill for some time before the fit, so that he not infrequently got into trouble in public places by assaulting the persons who felt it their duty to care for him when he seemed to require it. If let alone, he would pass through the seizure without harm, but the moment a hand was laid on him to assist him, he became extremely violent and would fight with great vigor until overpowered. He was always ignorant of his acts in this respect after his fit was over.

Fear, of a primary, instinctive, and therefore of an unreasoning kind, is the type met with in psychic aura, and it springs from the instinct of individual conservation.

According to Th. Ribot, in *The Psychology of the Emotions*, fear is the first, in chronological order, of the instincts to appear, showing itself, according to Pryer, at twenty-three days, according to Perez at two months, and according to Darwin at four months.

Ribot then goes on to speak of the characteristic marks of fear, so far as it affects the innervation of the voluntary muscles; of the muscles of organic life, and finally of the vasomotor apparatus, the concrete results making a very distinct picture of the condition of fear.

The very interesting question now arises as to how fear can be artificially induced; in other words, what is the process that brings it into existence so that it stands for a symptom in part of an epileptic convulsion? If we could answer this, we could place at least a part of our psychologic studies on firmer ground.

The warnings which take the form of "ideas" are generally dependent on special sense conceptions, especially on vision. Gowers mentions a woman who saw London in ruins, the Thames emptied to receive them, and herself the lonely survivor. This he calls a manifest "psycho-sensory warning."

All special disturbances such as these have the same basic meaning; they indicate some instability of the centers connected with the psychic processes.

I have referred to a woman who had an irregular aura, the first part of which was psychic and which gave her a feeling of great buoyancy and exhilaration. While this is not a common aura, it is distinct in type and of more than passing interest.

Binswanger, in describing the disturbances of feeling due to an approaching attack, states that "more rarely we find sensations of serenity, placidity, calmness, sudden apparitions of joy, rapture, colossal lightness, an elevating good feeling. Some patients declare that in the beginning of the attack they feel freed from a heavy burden." This exactly portrays the emotional state which the Russian novelist Dostoieffsky, who suffered from epilepsy, applies to himself in "Besi." "There are moments," he writes, "and it is only a matter of five or six seconds, when you suddenly feel the pressure of eternal harmony. This phenomena is neither terrestrial nor celestial, but is an indescribable something which man in his mortal body can scarcely endure. . . . The terrible thing is the frightful clearness with which it manifests itself and the rapture with which it fills you. During those few seconds I live a whole human existence."

Motor Aura.—It is often difficult to distinguish motorial forms of aura from the beginning of the convulsive period itself. Indeed, as we have previously stated, aura of all kinds might very properly be looked upon as the entering wedge of the fit itself, notwith-

standing that they may at times come and go without expanding into a convulsion.

A man, twenty-eight years of age, had a persistent motor aura in the form of a jerking movement that began in the right arm. It was just enough at first to lift the arm a few inches from his lap, but grew stronger each time, the jerks being two or three seconds apart, until finally the arm was raised at each impulse straight above the head. As soon as this condition was reached, the jerking began in the left arm, and followed the same course, until both arms were finally jerked over the head at each impulse, when a general convulsion ensued involving the whole body, the patient dropping unconscious to the floor. It required at times from half to three quarters of an hour for these motor manifestations to develop up to the convulsive point, a sufficient length of time in which to administer a remedy that often served to abort the attack.

A common form of motor aura is that of running,* just before the fit, the so-called *procursive epilepsy*. Patients who exhibit this phenomenon are rare, and, as a rule, are apt to be extremely violent during the attack. A boy would start and literally run like a deer for half a mile before falling in an active convulsion, which was always followed by great exhaustion.

Another case of the kind was that of a young married woman, who seemed to possess superhuman strength from the way she demolished the stoutest woodwork when in a *grand mal* seizure, and who could not be allowed out of the hospital for a walk because, on the point of an attack, she would run far enough away from the attendant to get lost.

When a seizure appeared while she was in the ward she would run at breakneck speed its entire length—

* Running in this instance is classed as an aura because it is a coördinate movement and readily separated from the fit that follows.

about 120 feet, jump up and thrust both legs through the window and out through the spaces between the iron bars, remaining in this position until the worst of the convulsion was over, it being almost impossible to tear her grip loose from the iron bars.

Gould and Pyle * mention "a peculiar case of epilepsy, the patient being a workman who would be suddenly seized with a paroxysm and unconsciously run some distance at full speed. On one occasion he ran from Peterboro to Whittlessy (distance not stated), when he was stopped and brought back, while on another occasion he ran into a pit containing six feet of water, from which he was rescued."

Echeverria † described the case of a girl twenty-two years old, who had general convulsions that always occurred in the daytime, the attacks being preceded by an uncontrollable desire to run. If held at the time and violently shaken, the paroxysms would occasionally be cut short.

These "running fits" came on at the age of twelve years and grew steadily worse, until the girl's mind was practically destroyed. As a remedy, it was directed that "she try violent exercise at skipping the rope, in addition to having a seton in the back of her neck, and being made to take large doses of the bromid of potassium and conium." The author adds that "the perturbation caused by the violent jumping evidently proved of great avail. No fits were repeated in the course of three weeks after beginning the above treatment joined to a tonic regimen."

The same writer mentions that violent exercise on a trapeze controlled the spasms in another patient; the patient resorting to the practice of his own accord in the belief that bodily fatigue would keep off the convulsions.

* "Anomalies and Curiosities of Medicine," p. 852.
† "Epilepsy," p. 257.

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A carpenter's son, a young man of twenty-four years, of powerful physique, often showed the first evidence of an attack by suddenly springing into the air, sometimes with such force as to turn a complete somersault, generally falling on the back and head; immediately afterward clonic contractions would set in, so violent as to require five or six strong men to hold him so that he would not injure himself; but as it was, his body for weeks after an attack was often covered with sores, bruises, and scars.

Other epileptics turn completely around one or more times before the attack comes on, the direction of turning being uniform; that is, if they turn to the left this time, they will turn to the left in subsequent attacks. Some walk backward a few steps, then fall heavily, striking on the occiput, repeating this often enough to keep an unhealed wound on the scalp at that point, unless a pad is worn to prevent it.

"Trembling from head to foot," as patients describe it, is witnessed in some cases, conjointly or not with an epigastric aura. A feeling of "giddiness" just before the fit is comparatively common.

Isolated contractions of certain parts of the body, like the thumb or fingers, or the great toe, may also announce the coming of an attack.

A woman who came under the writer's care a few years ago, began at one o'clock in the night to have a series of convulsions that were checked after the eighth one; but recurred six hours later and kept recurring for forty-nine hours and a half, until 549 had taken place. The initial spasm each time was a sharp, clear contraction of the first phalanx of the right thumb, as indicated in the illustrations (Plate 7). After two hundred seizures, the patient went into status epilepticus, the temperature gradually rising to 107°. Death occurred at the beginning of the third day.

Irregular Aura.—It is impossible to estimate with accuracy the frequency of irregular forms of aura, but they are not common, occurring less frequently than those of a psychic nature.

It was illustrated in a woman who first experienced a feeling of buoyancy and exhilaration which later gave way to a highly emotional state that persisted for several days, and sometimes even for weeks, before an attack; while for two or three days before the attacks a motor aura, in the way of uncertainty of gait appeared, the patient stumbling along as though half tipsy, or being unable to grasp or pick up a small object, such as a button or large needle; this grew more pronounced until the fit was over, when the bilateral paretic condition and unbalanced emotional state both completely disappeared. The irregular aura in this case was composed of psychic, sensory, and motor elements each dominating in turn, though being in part present at the same time.

Some have a warning of an attack in "a sleepy feeling," somnolence. It is impossible for them to keep normally awake. This feeling may exist for a day or so before the convulsion appears, being exactly opposite to another state in which the patient feels "unusually bright and active, mentally and physically," for some time before the fit, to be plunged into painful dejection after it is over.

H. N. Moyer* reports the case of a young man of good family history and correct personal habits until the age of fifteen, when he became alcoholic. At nineteen he was nervous, suffered from destructive diseases, and had hallucinations during his waking hours—seeing strangely formed animals approach him several times. In 1895 he became afflicted with a tendency to sleep, so that he had frequently to lie down several times a day for the purpose—from twenty

* "Medical Record," Nov. 19, 1899.

to fifty times the patient himself states—sleeping only a few moments each time.

When running an elevator he would sleep while passing several floors. He had other attacks which he described, which differed from the ordinary sleeping attacks. "Once at the table he suddenly became unconscious, the attack probably being epileptic."

It is difficult to say to what extent these attacks of somnolence may have been modified, psychic, epileptic convulsions. It seems evident that toward the last in the case above they were intimately associated with ordinary epilepsy.

In rare instances the heart is the seat of disturbances that indicate an approaching attack, some pain, but more particularly a "feeling of distress" being felt in the cardiac region, palpitation being comparatively common.

When *cephalic sensations* are experienced, they are generally dependent upon the extension of an aura that had its origin in some other part, like the stomach, though there is occasionally a sense of "fullness," or "a rushing sensation" experienced in the head or throat, independent of any disturbance anywhere else.

M. Allen Starr,* in a series of 65 cases of epilepsy, noted aura as follows: "Epigastric sensations were the most frequent, being present in 19; visual sensations of light, or sparks of blue or red in 14; tactile or sensory sensations in the form of tingling or numbness in the body or limbs in 10; in 7 the patients complained of vertigo; in 8 they experienced a sensation of fear without known cause; while in others there were palpitation of the heart, sudden inability to speak, a sensation of suffocation, or a feeling of drowsiness."

Church and Peterson† quote Lewis as having noted

* "Familiar Forms of Nervous Diseases," p. 258.
† "Nervous and Mental Diseases," p. 569.

an elevation of body-temperature during the aura, while Voisin has observed increased temperature in the limbs in which the aura originated. Church* obtained a sphygmographic tracing showing an increase in arterial tension before the fit, a fact also observed by others.

In a study of the temperature laws in epilepsy, made by the writer,† based on one thousand observations, to determine the degree of heat preceding or following convulsions of various kinds, it was possible to make but one satisfactory observation on the temperature any length of time before the fit, the aura in this case being epigastric and appearing two hours in advance. The temperature was normal when the aura first appeared, but immediately began to rise, and when the convulsion occurred two hours later, had reached 102° F.

Clark and Sharp‡ refer to the aura in forty cases of infantile cerebral palsy associated with epilepsy, as follows: "Fourteen patients had no aura, leaving twenty-six who had. In twenty of these it was sensory, in one psychic, one olfactory, one auditory, and in three visual.

"In eight of the sensory type, it is stated, the aura was confined to the paralyzed side, and was characterized as painful, the other sensory aura being distributed between epigastric, cephalic, and feelings of uneasiness.

"In one case, in which the epilepsy followed a paroxysm of whooping-cough, the aura appeared first in the nature of a typical *Globus hystericus*, and was followed by evidences of pharyngeal spasm similar to laryngismus stridulus."

It is now generally admitted that asthma is a vaso-

* "Nervous and Mental Diseases," p. 569.
† "The Medical News."
‡ "Pediatrics," Vol. VII, No. 7, 1899.

motor neurosis dependent upon irritation of the sympathetic nerve, the seat of the irritation, according to Glascow,* lying in the upper portion of the respiratory passages, including the posterior surfaces of the turbinates, the inter-arytenoidal commissure, the posterior surface of the trachea, and the membrane at the bifurcation of the trachea.

Bosworth† quotes Henry Hyde Salter as stating that "asthma is essentially, perhaps with the exception of a single class of cases, a nervous disease, the nerve centers being the seat of the essential pathologic condition," a proposition which, Bosworth adds, "is the one adopted at the present day."

Elsewhere we refer to certain asthmatic affections as constituting forms of "epileptic equivalents."

As a rule, the more sudden, complete, and severe the epileptic attack, the less likely it is to be preceded by an aura; while the further the attack departs from the classical type, the more frequent, persistent, and distinct the aura. At the present time we make but little use of these important initial manifestations. They justify close study as possible guides to the seat of the disease.

* "New York Medical Journal," Aug. 25, 1900.
† "Diseases of the Nose and Throat," p. 233.

CHAPTER IX.

SEQUELÆ OF EPILEPTIC CONVULSIONS.

The Effects and Results of Epileptic Convulsions. Proportion of Epileptics who Sustain Injuries. Tongue Scars. Scars of Scalp and Face. Burns. Hemorrhagic Extravasations. Fractures. Dislocations. Exhaustion Paralysis Body-temperature Other Effects, General and Specific.

BEFORE taking up the study of diagnosis proper, it will be well to briefly review the more common, marked phenomena that follow epileptic convulsions. Some of them are valuable from a diagnostic point of view.

The effects of the disease on the mind—its morbid psychology—will be studied separately.

In 825 cases (500 men and 325 women) 609 of them, equal to 74 per cent., had scars on some part of the body sustained as the direct results of epileptic convulsions. More men than women, by 10 per cent., presented these characteristics, a fact explained by the wider range of more hazardous vocations indulged in by men than by women.

TONGUE SCARS.

We customarily look for a scar on the tongue in every case, associating injuries of that organ with convulsions, while, as a matter of fact, they are more often absent than present.

Injuries of the tongue, particularly those of a minor nature received during an epileptic seizure, heal readily because of the tongue's vascularity and freedom from the ordinary sources of infection, and when not very severe, generally disappear in a few weeks, or a few months' time at the most.

It occasionally happens that the tongue is bitten

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through its thickest portion, or a piece is bitten from the side or tip. When this happens, the scar that follows may be permanent.

In 125 out of 500 men, and in 60 out of 325 women, there were one or more tongue scars at the time of examination, being about 22½ per cent. of the total number. It should be stated in this connection that when the search for scars was made, these 825 patients were having about 200 seizures a day, a fact which shows how recent most of the scars may have been.

I recall the case of a young man in which a severe glossitis followed an injury to the tongue during an attack, the tongue becoming so enormously swollen, firm, glistening, and white, as to greatly embarrass respiration, causing the patient great discomfort. This unique result of a fit may, in rare instances, justify the performance of tracheotomy.

Fischer* states that when this swollen condition of the tongue is aggravated by paralysis due to coma, death may be the result.

SCARS OF THE SCALP AND FACE.

Among the 825 patients 238 had scars about the scalp and face, the favorite localities for them being the forehead, nose, eyebrow, chin, cheek, and occiput, the ears occasionally suffering injury. Most of these were due to simple incised wounds that readily healed under proper care. The greatest danger to be feared from injuries to the scalp is erysipelas, a complication not infrequently met with.

FRACTURES AND DISLOCATIONS.

Among the same cases there were evidences of fractures in 29, most frequently the nose and clavicle being broken; the skull, legs, arms, and ribs occasionally.

* Fischer, 'Archives of Psychiatry," Vol. xxxvi, No. 2.

Plate 11.

A case of "alopecia epileptica," the bald spot developing coincidently with convulsions that followed a blow on the occiput.

Plate 12.

Illustrating a skeleton canvas jacket, devised by McBurney for use at the Craig Colony, to prevent dislocations of the shoulder during convulsions. Repeated dislocations of the shoulder occur during attacks in some patients. This device prevents them.

In the absence of any specific investigations, it cannot be said whether the bones of epileptics possess undue fragility or not. It is the writer's opinion, however, that in certain classes, notably the old, the rachitic young (in which the condition is to be expected), and in some cases of long standing in which the patient's stamina is thoroughly vitiated, the bones may break easily.

Reference is made in Tilton's "Text-book on Surgery" (p. 590) to fractures of the clavicle following the brandishing of a whip, or of the ribs in very old persons from coughing; while Scudder and Keen both speak of fractures of the patella as being "usually caused by muscular action," the result of powerful contraction of the quadriceps.

Fischer claims that contusions and fractures are found in 7.3 per cent. of all epileptics, which seems to me much too high. This author expresses the belief that the bones are unduly brittle in this disease, this condition resulting from "local or general trophic disturbances caused by the epilepsy or an antecedent nervous disorder."

Fractures caused by the force of muscular contraction alone during very severe attacks are possible. A male epileptic, thirty-eight years of age, mentioned to me by Dr. Rose, went to bed at night perfectly well, and the following morning had a fracture of the knee-cap and an intracapsular fracture of the femur, all evidences pointing to its having been received during a convulsion.

Dislocations during epileptic convulsions as the results of muscular contractions alone are not rare, eight such cases having come under my observation. In three of them the dislocations were almost habitual, occurring with each severe convulsion; in one the jaw was the seat of the dislocation; in the other two, the shoulder. Displacement of the latter occurred in

one case four or five times a month, until an apparatus, shown in the accompanying illustration, was devised at my request by Dr. Charles McBurney. When it was kept properly adjusted, no dislocation occurred.

Dislocations occurring during the fit can be best reduced, without an anesthetic, during the relaxed or comatose period which follows, and before the fit is over. If reduced later, an anesthetic must generally be used.

BURNS.

Burns are common in epilepsy, as shown by the fact that 104 out of 825 cases presented scars caused by falls into an open fire, on a stove, or against steam or hot-water heating-pipes or radiators.

Disfiguring and painful contraction of all the fingers follow burns caused by the patient grasping a pipe carrying steam or hot water, and convulsively holding to it until a burn to some extent of the third degree is received.

Severe burns about the face may be caused by the patient falling against a hot-water heater and remaining in contact with it some time, although the degree of heat in it may not exceed from 140° to 150° F.

The deformities that follow burns due to epilepsy are sufficient to make up a distinct group in themselves, while in certain instances they may be looked upon as undeniably stamping the victim as an epileptic. The illustrations presented in this connection show this point quite clearly. Such burns mostly occur on the hands, arms, and face, the parts most exposed, though no part of the body is exempt when the patient falls on a stove or an open fire.

HEMORRHAGIC EXTRAVASATIONS.

Extravasations of blood in the conjunctivæ during the severer forms of convulsions are by no means rare, seeming to occur more often in the outer portion of

Plate 13.

Severe burn of face causing loss of right ear, and the substitution by the patient of an artificial ear made by himself.

Plate 14.

Illustrating common forms of disfigurement in epilepsy, due to burns. In this case the beard covers the most unsightly scars about the mouth.

Plate 15.

Showing loss of fingers on one hand due to a burn received while in an
epileptic attack.

Plate 16.

Extensive burn of the face that threatened destruction of the eyesight,
caused by falling in the fire during an epileptic attack.

the conjunctivæ and having a tendency to spread, so that an injected spot half the size of a grain of corn in the beginning .may extend in two or three days until it almost covers the whole eye.

I do not recall having seen a case in which the hemorrhage occurred in the vessels within the eye. Gowers states that he has often searched for retinal hemorrhage in such cases, but always without result.

Hughlings-Jackson has described what he terms "retinal epilepsy," a condition of spasm in the retinal vessels during epileptic attacks which causes momentary blindness.

Punctiform hemorrhages covering one side of the face and neck are likewise not infrequent after *grand mal* attacks in certain individuals. This condition is noticed as soon as the fit is over, the face having a dark, diffusely mottled appearance, without elevation, the discoloration partly disappearing under pressure, to quickly return when it is removed.

As a rule, such extravasations are visible for some days afterward, first fading in changing colors like a bruise.

The fact that the face suffers most is probably due to the constricting band of clothing about the turgid neck, which increases the effects of the mechanical congestion.

When death follows a very severe convulsion, or a series of them, like status, similar punctiform hemorrhages are seen in the brain, more particularly in the white matter, in the pericardium, and in the membranes of the spinal cord.

Hare quotes Sandras* as recording a "*disposition singulaire et inexplicable*" in the skin of epileptics, who, when exposed to the sun become covered on the face and generally elsewhere on the body, with numerous "*taches rosées*," without any elevation, and which disappear when the person goes into the shade.

* "Epilepsy: Its Etiology and Pathology," p. 35.

I have never witnessed any such phenomena. The changing color, as described by Sandras, may have been due to rapidly occurring psychic attacks, the effects produced being due to changes in vascularity. It is not uncommon for epileptics of this type to have a seizure and "change color" from seventy-five to one hundred times or more in a single day.

Echeverria reports the case* of a woman of thirty years, who had a general petechial eruption on the face, neck, and limbs as the result of "*petit mal* attacks occurring in the daytime and spasms in the night," the former being "constantly succeeded by fits of laughter and mania." The eruption that came on after the nocturnal attacks was minute and confluent in character, and generally passed off within two or three days. The same writer mentions a similar case reported by Chatelet.

L. Pierce Clark† reports the case of a young woman, seventeen years of age, of a neurotic family, whose epilepsy followed scarlatinal nephritis at the seventh year, the attacks being largely motorial in character. She was accustomed to exhibit a profuse subcutaneous hemorrhage on the right side of the face and neck after each attack.

The discoloration remained very pronounced for two days, when it began to gradually fade out. This occurrence was apparently prevented from recurring by small doses of the fluid extract of ergot.

EXHAUSTION-PARALYSIS.

Exhaustion-paralysis, or a more or less complete but temporary loss of function of some part of the body may follow certain types of epileptic seizures. This condition of transient paralysis in epilepsy was first carefully observed by Bravais in 1824, as mentioned

* "Epilepsy," p. 284.
† "The Medical Record," March 26, 1898.

in his work on hemiplegic epilepsy. The condition was, however, known many years before to French investigators. Among those who have chiefly investigated the nature and cause of the state may be mentioned Todd, Robertson, Jackson, Gowers, and Féré. No new contribution to our knowledge of the phenomenon has appeared since the eighties, save the elaborate work of L. Pierce Clark.*

Some degree of paralysis, exhaustive in nature, is not rare after Jacksonian or partial epilepsy, and may be easily demonstrated in cases in which consciousness is retained during the fit; it probably occurs in some degree after every attack in this form of epilepsy. The fewer the muscles affected by the fit, the greater will be the subsequent weakness, inasmuch as it is a result of a local and complete discharge of one particular motor area. The phenomenon may occasionally follow Jacksonian attacks in idiopathic epilepsy in which the convulsion is confined to one side or extremity. The motor weakness more or less rapidly disappears after single fits, but when it results after serial or status periods in which the fits are partial in type and range, the paralysis may be fully as complete as that seen after an apoplectic stroke, and it may remain more or less marked for days or weeks.

In rare cases of idiopathic epilepsy the paralysis has been known to persist as an anomalous type of hemiplegia. The true nature of such permanent palsies is still a matter of doubt, although one of L. Pierce Clark's cases showed no evidence of a destructive lesion at the autopsy. Cases of permanent exhaustion-palsy must not be confused with old unrecognized palsies of infantile type, in which the lesion is made prominent once more as a result of epileptiform seizures, and upon which the seizures depend. The

* "Clinical Studies in Epilepsy," "Archives of Neurology and Psychopathology" Vol. II, 1899.

paralysis is always most marked in those parts which are engaged most in the convulsion. It is, therefore, an exhaustion of cortical elements; in other words, it is a problem in fatigue of cortical elements and not of muscles alone.

It is quite probable that the general exhaustion which follows general fits and which is covered by coma may be analogous to the local weakness here described. In rare instances a loss of function (sensory in type instead of motor) of some one of the special senses may occur; such as, for example, the sudden unilateral deafness or blindness on that side engaged most in the convulsion. The occurrence of a special sense loss without some evidence of convulsion or other symptoms of a seizure, should be counted of hysterical origin. Good observers, however, claim to have noted bodily paralysis without convulsion (Lowenfeld, Heidenhain, and Gowers). They believe the paralysis is the result of sudden inhibition of cortical motor elements. However true these observations may be, Clark has never observed it, nor have I been able to satisfy myself that some convulsion had not taken place in such cases. Undisputed evidence of inhibition paralysis in epilepsy can be afforded only when the hands of the observer are placed directly upon the muscles themselves, a rule for the study of convulsions of first importance and too little emphasized by many observers. It is extremely doubtful that any fit may occur without some motor manifestation.

We can generally diagnosticate the exhaustive nature of the palsy in epilepsy without a previous history by noting the absence of spasticity, atrophy, and vasomotor changes, all of which are common in old organic hemiplegics. If the patient has had a series of seizures and an old organic lesion also exists, we may assure the friends of the patient that the

increased paralysis will disappear in greater part in a few days after the cessation of the seizures.

The illustrations presented in conjunction with two cases show the commoner forms of exhaustion paralysis:

Case I.—F. R., a man aged fifty-three years; iron-worker by occupation; age at onset of epilepsy, fifty years. The cause was ascribed to grippe, but the patient has undoubtedly used alcohol to great excess. No neurotic history was obtainable. His seizures were not preceded by any aura. The reflexes, both superficial and deep, were normal on admission, March 25, 1896. There was a slight muscular tremor in the hands and lips.

On April 6th he began to have an average of twelve seizures in twenty-four hours. After the first two days these attacks were followed by a partial hemiplegia of the right side; after the first six days he was able to move his fingers only. The epileptic seizures were always of a general convulsive character. For the first few days, and whenever these seizures would cease for two or three hours, he could perform some of the very simple movements of the arm and leg of the affected side. Fig. 11,* pose of F. R., shows the patient with the seizure involving the left side first and beginning to involve the right side of head. Fig. 12 of the same convulsion shows the seizure finally ending on the right side in the extreme tonic convulsion, proving the right side to be the one most involved in point of intensity. Fig. 13 shows the same patient some days later in convulsion with the right side put entirely out of action, as it were, because of the apparently complete exhaustion of that side remaining from previous attacks like those shown in Figs. 11 and 12. The left side alone remained capable of carrying on the convulsion phenomenon in subsequent seizures. The deep reflexes of the right side were very much exaggerated; all through his series of attacks ankle-clonus was a prominent symptom. Plantar reflex was always

* The negatives taken from the actual convulsion were too faint to allow of reproduction, but were used as guides in these poses (of Figs. 11, 12, and 13) which are particularly accurate.

obtainable and was markedly exaggerated toward the end of the series.

During the nineteen days following the first attack he had 249 distinct and separate convulsive seizures.

On April 21st he began to have fewer attacks daily, and on the 25th he had only one; for the next eight

Fig. 11.
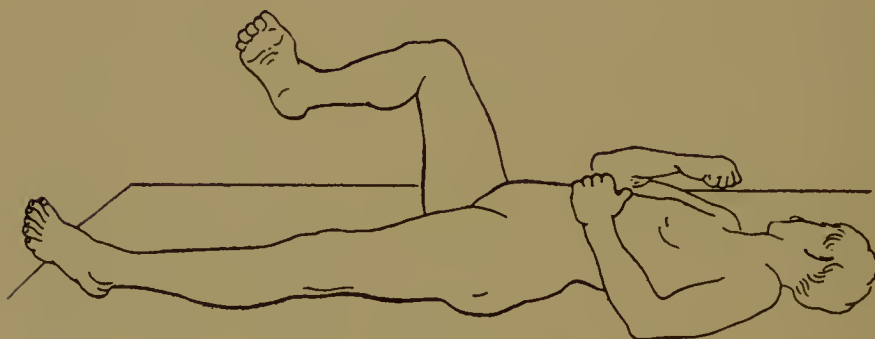
Fig. 12.
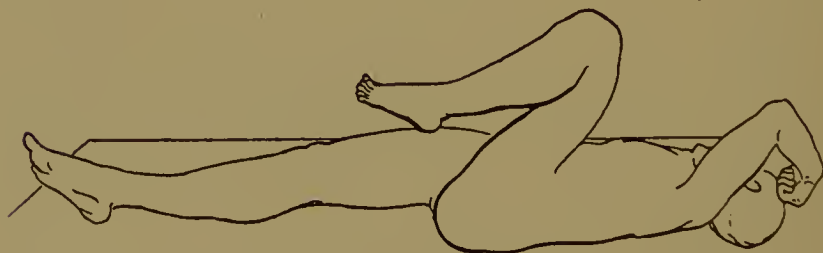
Fig. 13.

days he had but one attack a day. At the time he began to have but three attacks a day he slowly picked up some of the less complex and comparatively simple muscular movements. Throughout the entire period the superficial and deep reflexes remained exaggerated and were much more noticeable on the

affected side. A small corneal ulcer made its appearance on the 24th, and a bedsore began on the right buttock the next day. Both were healed very quickly by local application. During this time the patient lost twenty pounds in weight. From April 24th to May 15th he made uninterrupted progress toward recovery of almost all of the muscular movements, but there still remained some slight paralysis of the more complex muscular movements. The deep reflexes of the left side at first were less than the right, and below a normal reflex. This condition persisted until the stated improvement began; then they became exaggerated equally with the right side.

The patient's condition six months after his paralysis was as follows: The knee-jerks of both sides were exaggerated, the right more than the left; ankle-clonus was still obtainable in both sides, although more marked on the right; both wrist-jerks were markedly exaggerated, the right greater than the left. The right pupil was incoördinate in its adaptation to light, and the patient was incoördinate in his attempts to perform many of the more complex muscular acts involving muscles of the affected side. He stated that he was "very awkward and clumsy."

An examination made in April, 1898 (two years after his attack), showed complete recovery of all the paralyses. Several attacks occurring in series since, attended by temporary paralysis, prove the exhaustive nature of the resulting paralyses. The patient is at present (October 1, 1899) doing a full day's work at his trade (that of a blacksmith), having occasional isolated epileptic fits.*

Case II.—M. E. H., a girl aged twelve years. Epilepsy began at four years and a half. Neurotic family history. Seizures dated from infantile cerebral palsy with left hemiplegia. During the first two years of epilepsy the convulsions were confined to the left side, but later became general. There were occasional

* Note by the author: June, 1903. The patient's condition is more satisfactory now than when the last note was made in 1899. He is robust and hearty in every respect, showing no sign of exhaustion-paralysis in any degree. During 1900 he had 145 seizures, during 1901, 59, and during 1902, 66; none of them being followed by the condition described above.

petit mal attaeks.   There was a loss of speech with left side palsy.   The patient reeovered suffieient power in the hemiplegie side to walk to some extent after five months from the onset of the palsy.   At various periods during the past two years the patient had a series of eight or ten eonvulsions in forty-eight hours, the left side being chiefly involved.   An after-palsy nearly eomplete lasted from one to two days.   Atrophy was most marked distally; there was but little eontraeture.   Extremities of both sides were equal in length.   All reflexes of the left side were exaggerated.

In July, 1898, typieal exhaustion paralysis supervened upon the left (palsied) side, leaving that side completely hemiplegie for weeks.   The right side remained unaffeeted.   Seizures convulsed the left side most, beginning and ending on the left side.   (See Fig. 14.)

The exhaustive phenomenon of the right side and arm was first manifest in the series of attaeks from September 28th to Oetober 8, 1898.   The temperature on September 30th reaehed 106° F., pulse 168, respiration 60; two hundred and sixty-three seizures oeeurred that day, mainly loealized in the right side and predominating in the right arm.   This is admirably shown in Text-figure 15 drawn from the photograph.   The left arm remained free from eonvulsion.

On Oetober 8, 1898, the fingers of the right hand assumed a state of athetoid spasm (Strümpell); the

Fig. 14.   Fig. 15.

⫽ indicates slight convulsion.
∷ indicates slight paralysis.
♯ indicates severe convulsion.
✕ indicates severe paralysis.

index finger was extended; the thumb was flexed to the side of the metacarpal phalangeal joint, and over the third and little fingers in semi-flexion at the second joint. The third and little fingers were flexed only at the knuckle; other joints of the fingers were nearly straight. Continuous slight fibrillary tremor was found in all fingers, especially the third and little fingers. Earnest voluntary effort enabled the fingers to be partially extended at the knuckles, but such effort was attended by some pain and performed very slowly. Painful contracture which complicates palsy is called by some the post-epileptic paralytic equivalent.

On December 14th paralysis continued most marked in the right upper extremity. The right hand at rest frequently assumed Strümpell's typical athetoid position. The movements of the fingers were mostly those of mobile spasm; no tremor was noticed; there was much less contracture than formerly. Upon strong volitional effort, considerable power was manifest in the hand. The right shoulder and arm muscles still appeared weak. The extremity, as a whole, was lifted very slowly and with great effort. The right hand was cold and the circulation was very poor.

On January 8th severe convulsions occurred at 8.45 A. M. and 3.25 P. M., both similar in character, and affecting the left side most, although apparently general. This was followed by carpopedal contractures, beginning immediately after convulsion and continuing for thirteen minutes, gradually diminishing in severity toward the last. During the first six minutes the left arm and right leg were most affected; afterward the right side alone was involved. The toes of the right foot were in hyperextension, the foot inclined inward from the ankle; the leg was flexed at the knee (this position was taken suddenly, the leg being immediately straightened when contracture subsided). The right hand was semiflexed and the wrist completely flexed, the arm remaining straight.

A slight convulsion occurred at 3.00 P. M., followed by carpopedal contractures affecting the left side most; it continued for ten minutes. There was slight weakening of the left side: the right side remained unchanged.

On February 10, 1899, the patient had two kinds of paralysis. The left side had flaccid, soft, and atrophied muscles, giving a spastic gait; the right side was in slight rigidity and contracture, giving ataxic gait. This condition showed constant improvement.

The degree of the paralysis of the left side is dependent on the severity and frequency of the attacks. Cessation of attacks for a few days allows the side to return to the normal inter-paroxysmal paretic state. Although in severe series the convulsions are confined mostly to the left side at first, they change to the right side almost entirely. They apparently do not do this until there is complete paralysis of the left side. Exhaustion-paralysis then rests upon an organic paralysis, producing complete paralysis.

The cerebral center of the right side, being left alone to carry on the convulsive state, is soon exhausted and becomes more or less paralytic, but will recover again more or less completely if allowed to rest for any marked period.

BODY-TEMPERATURE.

Bourneville, assisted by Lemoine, was the first to study the question of increased temperature following an epileptic convulsion. His earlier studies dated from 1869 to 1886, while he made a second series of observations extending from the latter year to 1900. After his first work, the results given by him were repudiated by Witkowski, which led Bourneville to repeat all he had done in a most careful manner, presenting conclusive evidence on every point.

So satisfactorily did Bourneville do his work in the first instance that Charcot and other eminent teachers of the day dogmatically taught that there was a rise of temperature after epileptic convulsions.

Bourneville's and Lemoine's work was based upon over 500 examinations. Of these Bourneville reported

64 cases in 1886, but it is not yet apparent when these investigations were made. In his "Recherches sur l'épilepsie," etc., for 1886–1887, he reports 109 cases. In the same "Recherches for 1891" he reports 66 observations, none of which were made prior to 1888. Lemoine in 1888 reports 182 cases. The grand total is 520 cases.

As a general rule, the majority of these observations follow a law and may be described as typical, while a small minority are atypical. Of the first series of 64, 60 were typical; of the second series of 109, 82 were typical; of the 66 cases, 58 were typical. Lemoine, who considered the subject from a broader standpoint, found in his 182 cases only a solitary exception to his normal, or type. Lemoine assumed that certain cases were naturally anomalous in the matter of temperature, having abnormally low temperature in health; hence the anomaly in these cases is not referable to the epilepsy, but to the patient himself. From Bourneville's standpoint there would be about 92 per cent. typical, and from Lemoine's point of view upward of 99 per cent.

Isolated Attacks.—Bourneville's conclusions were as follows: (1) Isolated attacks of epilepsy augment the central temperature; (2) this augmentation varies from .1° up to 1.5° and even higher at times. The average rise is about .5° or .6°.

Serial Attacks.—In Bourneville's article, published in 1891, he gives his observations on four cases of serial attacks in which he took three temperatures as follows: The first at a given time, the second twelve hours later, and the third eighteen hours later. Of these four cases there were respectively five, fifteen, twenty, and nine attacks in one day. The first temperature was taken at the moment of stertor after an access. In all the cases the initial temperature was the highest, and the readings at twelve and eighteen hours were

progressively lower. The initial temperature varied from 38° to 38.6° C.; the twelve-hour temperature from 37.8° to 38° C., and the eighteen-hour temperature from 37.3° to 37.8° C. It would appear, therefore, that these cases do not differ materially from ordinary isolated cases.

Status Epilepticus.—This clinical phenomenon was first clearly described by Bourneville. He characterized it as follows: (1) Attacks incessantly repeated so that they overlap one another (subintrant attacks); (2) terminate in collapse variable in degrees up to the most absolute and fatal coma; (3) may end in hemiplegia; (4) pulse and respiration always increased; (5) considerable elevation of temperature, which persists during the (brief) intervals between convulsions and increases even when the convulsions have ceased.

Bourneville gives several fatal cases in which the temperature ran up respectively to 40.8°, 42°, 42.4°, and 40.9° C. In 1892 Bourneville reported a similar case in which the temperature was taken up at death. The record shows that the temperature increased slightly each day until at death it was 40.2° C.

Intermediate Cases.—Bourneville reports two cases which he considers stand midway between ordinary serial attacks and the status epilepticus. These are given in his "Recherches sur l'épilepsie," etc., 1891, but are not especially significant, being thoroughly unique and atypical.

Personal Observations.—My own studies of the temperature laws in epilepsy, made in 1901,* extended over fifteen months and covered 1000 records made in 250 cases. These included all types of the disease, though in some of them, such as the so-called hysteroepilepsy, a sufficient number of observations could not be made to warrant any general deductions being drawn.

* "A Study of the Temperature Laws in Epilepsy based on One Thousand Observations," "Medical News," September 7, 1901.

The 1000 observations were divided into seven groups, as follows:

Group   I. 516 *grand mal* seizures.

Group   II. 133 *petit mal* seizures.

Group III. 81 psychic seizures.

Group IV. 50 *grand mal* seizures with extended observations.

Group   V. 25 *petit mal* seizures with extended observations.

Group VI. 17 cases of status epilepticus.

Group VII. 5 cases of serial attacks.

GROUP I.—This group comprised 516 *grand mal* seizures. The average duration of all the seizures, including the stage of active convulsions only, was a fraction over one minute and three quarters, the longest being five minutes, the shortest half a minute. A single fit lasted only five minutes with a temperature of 99.6° F. immediately after, dropping to normal thirty minutes later. In five cases the fit lasted four minutes, all followed immediately after by increased temperatures, ranging from 99.2° to 100.6° F. In ten others, in which the fit lasted half a minute only, the temperature was increased in all from .2° to 3.5°. This fact is mentioned to show the apparent lack of value in comparison between heat elevation following seizures of long duration, as compared with those of short duration.

Taking the 516 *grand mal* seizures collectively, it will be seen that 297 (57½ per cent.) showed an increase in temperature ranging from .2° to 3.5° immediately after the fit. Again, taking the same group of cases, it is seen that 80 (15½ per cent.) showed subnormal temperatures immediately after the fit, ranging from 96.4° F. in one case to from 97° to 98° F. in thirty-one others. As this subnormal group will not be referred to again, it may be well to state here that, after careful study, I have been unable to assign any cause

for the low temperature in these cases, other than the causes that operate generally to produce low temperatures in other conditions, such as chronic asthenic diseases of long standing, defective nutrition, idiocy, imbecility, and epileptic dementia complicated with or without paraplegic or hemiplegic infirmities.

Turning again to the 516 *grand mal* seizures, it will be noted that while 297 (57½ per cent.) showed increased temperature immediately after the fit, 330 (66 per cent.) showed an increase half an hour later, the highest temperature being 102.2° F.; the lowest, 97.4° F. At the third observation, an hour after the fit, 327 (64 per cent.) gave temperature increases above the normal, ranging from .2° to 3.5°.

GROUP II.—The average duration of the fit in this group was one and two-tenths of a minute, the longest being four minutes, the shortest fifteen seconds.

Immediately after the fit 80 (60 per cent.) showed increased temperatures ranging from .2° to 2.5° above normal, while a subnormal record was made in 14 (10 per cent. of the entire number), the proportion of subnormals being less in this group by 33 per cent. than in the previous *grand mal* group. The same 133 cases presented at the end of half an hour increased temperatures in 94, or 70 per cent., ranging from .2° to 4.5° above normal, while at the end of an hour after the fit only 70 (52½ per cent.) still showed an increase.

GROUP III.—The average duration of the fit in this group was about one minute. Immediately after the fit 49 (60 per cent.) gave increased temperatures ranging from .2° to 1.8° above normal. After half an hour 56 (69 per cent.) still showed an increase; while at the end of an hour 54 (67⅓ per cent.) showed relatively the same heat elevations.

GROUP IV.—In this group, 26 (52 per cent.) showed temperature increases that ran uniformly through

all the record hours; that is, those showing an increase immediately after the fit showed relatively the same increase at the end of the second hour. It may also be noted that 14 cases of this group, which showed low temperatures immediately after the fit, showed still lower temperatures two hours later, many of them dropping from slight increases above normal to sub-normal.

GROUP V.—In this group 13 (52 per cent.) showed increased temperatures immediately after the fit. An hour after 16 (64 per cent.) showed increase; while at the end of the second hour after the fit 17 (68 per cent.) showed relatively greater increases than were noted immediately after the fit.

None of the seizures of this group were violent or severe, none lasting more than sixty seconds, so that in looking for the cause of heat elevation in them we feel justified in at once excluding muscular activity. In many of them the muscles did not play any more important part than that required for the fibrillary contractions of some of the muscles of expression, chiefly those about the mouth, which are so frequently seen in *petit mal* and psychic seizures.

It is not possible for such muscular activity to increase the temperature of the body to 100° F.; and in such cases it is entirely within reason to assume that there has been a disturbance in the central nervous system, either in the thermal cortical centers described by Landois and Stirling, or, since motion in such cases is so significant, in the lower heat centers in the corpus striatum and optic thalamus.

It is a well-established fact in pathology that in disease involving the highest specialized tissues of the body, recovery takes place at a much slower rate than when grosser tissue is involved. By analogy, the same thing is true here. Increased heat of the body, due to simple increase in muscular action, will more

quickly pass away than when such increase is due to a deep-seated disturbance of the heat centers of the brain.

GROUP VI.—The fact that status epilepticus is so often accompanied by high temperature is so well known that further arguments on this line would be superfluous. This group is presented more to make the study of the temperature laws in epilepsy as complete as possible than for any other purpose. The lowest status temperature was 102.4° F.; the highest, 107.5°. In all the temperature ran up to 104° F. and over, and in nine cases to 105° F. and over; while 107° F. and over was reached in two cases.

GROUP VII.—The difference between the duration of the attacks in this group, as compared with the duration in the status group, and further compared as to the temperatures that followed in each, is apparent at a glance. The highest temperature following serial attacks was 105.8° F., the seizures that caused it numbering 140, covering a period of ninety-six hours. In none of the remaining serial attacks did the temperature go above 100.8° F.

Death occurred in 28½ per cent. of the status cases; deaths occurred during serial attacks.

In his conclusions Bourneville says: "Isolated attacks of epilepsy augment the central temperature." The only logical inference we can draw from this statement is that Bourneville found the temperature increased after every isolated epileptic seizure, and nothing exists in Bourneville's work on the subject to change this opinion.

The results of my own observations agree in part only with those of Bourneville, the latter's work having, in my opinion, lost much of its value through failure to classify the types of seizures studied and to make due allowance for certain physiologic differences in temperature.

After making due allowance for diurnal variation,

it will be noted that 40 per cent. would be the lowest of such cases showing increased temperature after seizures, and 70 per cent. the highest, making the general average of cases of all types showing increased temperature after seizures 55 per cent., the difference in the results obtained by Bourneville and by the writer being 45 per cent. The views of such eminent physiologists as Dalton, Kirk, Foster, Landois, and Stirling, relative to the part played by muscular activity in creating heat, must in view of the activity of the muscular system in certain cases of major epilepsy have great weight in determining the cause of the heat production in these cases, wholly irrespective of any influence that might be exerted by the heat centers in the brain.

By the results of my own observations, I am led to believe that in many *petit mal* and psychic attacks in which muscular activity plays so small a part, and in which the temperature is often increased after seizures, such increase is due to a disturbance either of the heat center that is thought to exist in the cortex of the brain, or of the center or centers that observers believe have been located in the corpus striatum and optic thalamus.

It will be noted that subnormal temperatures followed epileptic seizures in greater proportion after *grand mal* than after *petit mal* or psychic seizures, the proportion being 15 per cent. of the former to 10 per cent. of the latter, and while I agree in the main with Lemoine as to the cause for such low temperatures, when he says: "Certain cases are naturally anomalous in the matter of temperature, having abnormally low temperatures in health," I do not believe that this explanation is altogether sufficient, but that there will usually be found to exist in these cases some chronic disease or general asthenic condition of long standing that lowers the stamina and vitality of the individual.

All that has been said by Bourneville, Lemoine, and others, relative to the high temperaturc in status epilepticus, is confirmed by the tabulated results in the seventeen cases studied by the writcr.

The temperature in serial attacks runs uniformly higher than in isolated attacks, but not so high as in status, serial attacks occupying midway ground as to frequency, severity, temperature elevations, and mortality, between isolated seizures and status epilepticus.

As a possible factor in establishing the presence of toxins or other agencies in the body prior to and possibly causing the convulsions, an effort was made to take the temperature in some cases before the attack when the aura was of sufficient length; but it was done in one case only, and in this the heat, beginning two hours before the fit, ran steadily up to 102° F., when the patient passed into a convulsion.

Notwithstanding the apparent lack of value of the feature of temperature studies in epilepsy, its value in differential diagnosis, as we shall see later on, may be considerable.

OTHER EFFECTS OF EPILEPTIC SEIZURES.

Effects on the Eye.—Among the more specific effects of epileptic convulsions, dilatation of the pupils occupies a prominent place. This is especially true of the cortical epilepsies in which muscular contraction is a feature.

I fail to recall a single genuine epileptic convulsion witnessed in which both pupils were not more or less dilated—the dilatation in somc instances being extreme. Two reasons are assigned for this: one, that it is due to the effects of asphyxiation; the othcr, that it is due to the direct effects of the convulsion independent of asphyxiation. Probably both rcasons are correct in part.

It does not seem that asphyxia can be the one cause in all cases. I have personally witnessed numbers of attacks light in character in which there was absolutely no evidence of interference with respiration, yet in which mydriasis was marked early in the seizure.

I recall several cases in which I happened to have my hands on both wrists of the patient. The fit began by gentle contractions in the muscles of the hand and arm, and I observed that dilatation of the pupils appeared before there was any muscular evidence of the fit whatever; nor was there in any of these cases any fixing of the chest muscles so as to cause asphyxia in this way. This would seem to indicate an irritative disturbance in the central nervous ocular apparatus separate and distinct from the respiratory center.

The effect of the attack on the pupils furnishes a diagnostic point of value in differentiating epileptic convulsions from those of hysterical origin.

Equal dilatation is the rule in inorganic epilepsies in which the cause is in the cerebrum.

If the epilepsy is due to local organic conditions in the brain, the dilatation may be more marked on one side than on the other, not only during the attack but during the interim.

As a rule, the corneal reflex is abolished during epileptic convulsions, especially when the attack is severe and complete loss of consciousness is an early indication. In rare cases in which the attack is rudimentary in character, the cornea retains its sensitiveness to some extent throughout the fit—paralleling the effects of hysteria in this respect.

I have previously spoken of what is termed *retinal epilepsy*, a form of attack made up by sudden blindness of brief duration—*epileptic amaurosis* it is called by Hughlings-Jackson.

Hemorrhagic extravasations into the conjunctivæ

during an attack have also been mentioned. These at times constitute the only visible sign on the morning following a nocturnal attack, though they are more apt to be present in conjunction with other indications, such as a sore tongue, headache, malaise, etc.

Effects on the Blood.—Chemical pathology is not yet a sufficiently elaborated science to enable us to detect blood changes in epilepsy, that in truth may antedate and possibly cause the convulsion, as well as appear as the result of the fit. At this time little is known of either.

The researches of Herter along this line have been most valuable, embracing the study of the toxicity of the blood in fifteen cases at the Craig Colony.*

In these instances, Herter tested the toxicity of the blood after *grand mal* seizures and again after fifteen, sixteen and a half, and twenty-six hours respectively. In one case the blood appeared more toxic at the second bleeding than at the first; in another, more toxic at the first bleeding than at the second; while in the third it was about equally toxic on the two occasions.

Blood drawn in another case three weeks after a seizure, and two weeks before another, and infused into a dog had no effects other than those due to the effusion of normal blood in large volume. In a second case, blood drawn fifteen days after a seizure and several days before another was infused into a Java monkey weighing 1200 grams, 145 c.c. being introduced. The effects were those expected to follow a large volume of normal human blood thrown into the circulation, death being referable to the mechanical action of the infused blood. Defibrinated blood was used and the rate of infusion uniformly 5 c.c. per minute.

* "Toxic Properties of the Blood in Epilepsy," "Journal of Nervous and Mental Diseases," Vol. xxvi, p. 73, 1899.

Herter refers to the scope and limitations of such investigations as being great, while the chance for error is also great; and he gives suggestions of value to those who undertake further researches along a similar line.

Effects on the Circulation.—In 284 cases in which the pulse beat was noted, it was found to run uniformly above the normal in 189, 64 per cent. In 20 cases it was below 70; in 74 cases, from 70 to 80; in 161 cases, from 80 to 100; while in the remaining 29 cases it was 100 or over.

It was counted in every instance during the inter-paroxysmal period, the patient being entirely free from any immediate epileptic influence. The cause of this apparent phenomenon is uncertain.

A rapid heart is seen most commonly as a result of stimulation by drugs, by fear, or through some form of fever. None of these is applicable in this case, except, possibly, in rare instances. The explanation may eventually be found, in part at least, in the repeated and powerful depression of the pneumogastric nerve.

In the study of thirty-three cases to determine the relative blood-pressure as near to the convulsive period as possible, and as remote from the same period as possible, the results given in the table on page 272 were noted.

To summarize, there may be seen in this table an apparent decrease in the blood-pressure just before the convulsive period in eleven cases, and an increase in twenty-one at the same period, the latter being somewhat more marked in degree than the former.

Thus it may be noted that in a number in which the reading before the fit ran from 135 to 175, twelve hours later it had dropped from 20 to 60 points; in one, 15; in one, 30; in two, 35; in one, 45; in one, 55; and so on. This should not lead us to suppose that increased

blood-pressure was the cause of the attacks, for it is yet to be proved that such pressure was not the result, in part at least, of the convulsion and not the cause.

On the other hand, we find the pressure quite often decreased before the fit, a fact that may be as significant as the former; in fact, in the writer's opinion, it is not infrequently more so.

BLOOD PRESSURE RECORDS IN THIRTY-THREE CASES BEFORE AND AFTER THE CONVULSIVE PERIOD.

Age.	Hour at which the Attacks are most Apt to Occur.		Hour at which the Blood-pressure is Taken.		Pressure Record before the Convulsive Period.	Pressure Record at the Hour most Remote from the Seizure.	Normal Pulse-rate.	Pulse-rate at the Convulsive Period.
18	7 to	9 p.m.	12 hours later		105	110	78	80
14	"	"	"	"	95	90	96	102
25	"	"	"	"	140	120	84	108
18	"	"	"	"	115	120	78	66
26	"	"	"	"	135	100	72	66
21	"	"	"	"	105	125	66	90
14	"	"	"	"	90	145	90	96
18	"	"	"	"	100	100	90	78
15	"	"	"	"	95	90	90	66
10	"	"	"	"	85	80	108	96
18	"	"	"	"	105	115	66	72
13	"	"	"	"	110	105	120	108
39	"	"	"	"	150	130	84	72
20	"	"	"	"	120	100	122	90
18	"	"	"	"	130	125	72	84
18	"	"	"	"	160	115	84	66
28	"	"	"	"	125	120	120	90
18	9 to	10 p.m.	"	"	108	120	88	68
26	"	"	"	"	135	115	84	76
43	"	"	"	"	155	155	96	100
24	"	"	"	"	135	130	80	76
22	"	"	"	"	120	100	88	88
32	"	"	"	"	125	110	76	88
42	"	"	"	"	150	115	80	76
25	"	"	"	"	175	120	104	68
25	"	"	"	"	140	145	76	64
38	"	"	"	"	144	155	88	88
24	"	"	"	"	95	105	80	88
29	"	"	"	"	105	90	76	84
25	"	"	"	"	130	115	96	88
40	"	"	"	"	155	135	76	88
34	"	"	"	"	105	110	88	76
17	"	"	"	"	130	105	100	76

From carefully recorded observations in 58,873 seizures in one group of cases at the Craig Colony in 1902, the greatest number occurring at any one hour was 3628, the hour being 3 A.M., the time at which the vitality of the body reaches its lowest ebb.

A girl of thirteen years, whose epilepsy followed a severe, acute attack of enterocolitis in her sixth year, and who never had an attack during the period of two years she was under my observation, save when she slept either by night or by day, averaging from three to five or more seizures each night, showed a blood-pressure of 95 just before 9 P.M., the first convulsive period of the night; while during the day following it was from 125 to 130.

It is evident to the writer that low pressures are the rule in some cases before the fit, while relatively high pressures are the rule in others.

The diagnostic value of blood-pressure in epilepsy is as yet even more in its infancy than in other conditions. It is a promising field for investigation.

CHAPTER X.

DIAGNOSIS.

The Facies Epileptica. The Distinguishing Features between Epilepsy and Hysteria. Alcoholism. General Paresis. Syncope. Toxemia. Vertigo. Uremia. Tetanus. Simulation.

I HAVE already so fully described the symptoms of epilepsy that differential diagnosis only will be considered in this connection.

The diagnosis of epilepsy as a rule is not difficult. If the fit can be witnessed by the physician from the moment the first symptom appears until the coma stage is over, there is scarcely a case in which there need be any question as to its nature. Without this, errors are always possible, though the proportion of cases that create diagnostic uncertainty are rare, representing not more than one or two per cent. at most.

The only way to guard against errors in this respect is to acquire clear conceptions of the numerous forms of the disease through the study of actual cases.

The information we get from the patient or his friends is not infrequently misleading, and it is not safe to base a diagnosis on it in all cases. Especially are we unable to rely on such information as to the type of the disease, which is often as important as the fact of its presence. Only a trained observer can make such distinctions.

The most satisfactory plan is to place the patient under the care of a skilled attendant who knows how and what to observe, requiring a minute record to be kept of every convulsion until the physician can fully satisfy himself as to the exact nature of the disease.

The record of attacks should include the aura, its nature and duration; the order of invasion, the part of the body in which the convulsion began, the order of extension; the part of the body or group of muscles affected one after the other; the stage of the convulsion at which consciousness was impaired or lost; and if impaired only, to what extent; the condition of the pupils; the duration of the period of tonic contraction, of the period of clonic contraction, together with that of coma which so often follows. After-effects should also be noted, such as mental disturbance, headache, hemorrhagic extravasations, irritability, thirst, exhaustion, post-epileptic paralysis, muscular soreness—general or local—together with any injury or loss of function, temporary or more lasting, occasioned by the attack.

The Facies Epileptica.—The condition sometimes observed in epilepsy and termed the *facies epileptica* has a very limited diagnostic value, for the reason that it is never present save in chronic cases in which the identity of the affection is already fully established.

Facial expression is produced by the formation of creases, changes in the contour of the skin and underlying structures, due to muscular and trophic alterations, to the patient's habits of mind, his intellectual standard and temperament; and in disease it may be changed by pathologic processes that affect other and remote parts of the body.

Epilepsy may affect the face directly or indirectly in several ways: (*a*) Indirectly by covering it through medication with bromic acne which is more commonly associated with epilepsy than any other disease; (*b*) by so altering the patient's mentality in many cases as to make the reduction in intelligence noticeable in the face. The countenance of the epileptic dement is especially heavy, sodden, and devoid of every aspect of mind; (*c*) through the infliction of scars, fractures

of the nose and jaw, burns, bruises, incised wounds, and other injuries received during convulsions.

The true facies epileptica, the only form possessing any distinctive worth from a diagnostic standpoint, is that made up of the trinity of results, namely, epileptic dementia, scars, and bromic acne. These together produce a facial condition that cannot be mistaken, but being a composite picture of slow formation, is of little or no value in the diagnosis of recent epilepsy.

Hysteria.—The one convulsive disorder more apt to be mistaken for epilepsy than any other is *hysteria*. The statement of M. Briquet,* that "hysterical subjects are apt to be seized from time to time with special and serious conditions which appear of a sudden and usually, after a short duration, disappear as quickly as they came," illustrates the points of similarity in a general way between the two affections—both are special and apparently serious conditions at the moment, and both repeatedly appear and disappear with great suddenness. The hysteric state, independent of convulsive attacks, has but little interest for us in this connection. The ground for confusion is that occupied by the attacks alone. We occasionally meet with cases in which the convulsions of hysteria alternate with the convulsions of true epilepsy, the attacks being epileptic to-day and hysteric to-morrow. Under such circumstances, the diagnostic view-point is necessarily altered—taking its color from the type of the condition present at the moment.

This makes it desirable for the physician, in the more doubtful cases at least, not to be hasty in giving his opinion, but to wait until he has opportunity for witnessing several seizures, or of having their nature reported to him by a competent observer.

In my experience, men a little more often than

* Janet, "The Mental State of Hystericals," 1901, p. 366.

Plate 17.

Accumulated scars on supraorbital ridges, nose, and other parts of face, received during epileptic attacks. Such scars constitute a part of the so-called "facies epileptica."

women have hystero-epilepsy, while purely hysterical convulsions are commoner among women.

TABLE SHOWING MAIN DIAGNOSTIC POINTS BETWEEN THE CONVULSIONS OF HYSTERIA AND EPILEPSY.

	Epilepsy.	Hysteria.
Age	Common before puberty and rare as a recent affection after the twentieth year.	Very uncommon before puberty.
Exciting cause	Rarely due to emotional disturbance after the disease is established.	Often due to emotional causes.
Warning of attacks	May be of any form. Generally affects a part of the body first, or one of the special senses, or the aura is epigastric.	Generally preceded by palpitation; a feeling of malaise, choking sensation; special senses not affected.
Mode of onset	Most apt to be sudden. Epileptic cry quite frequent, and is usually sharp and distinct, the patient falling heavily to the ground unconscious.	May be gradual, the cry long drawn out, the fall easy, and rarely with injury to the patient—injury never as severe as in epilepsy.
Type of convulsion	Usually begins locally with tonic contractions followed by clonic or "jerking" movements; rarely rigidity alone.	May be simple rigidity, or violent general struggling. No clonic movements. The patient is tossed about from side to side, the limbs thrown wildly about in the air, the head turning from side to side.
Consciousness	In the majority of cases, lost; always in the classical *grand mal* attacks.	Impaired only or fully retained.
Duration of convulsion	On an average from one-half to two and a half minutes for the active period only, not including the periods of stertor and coma.	Several minutes up to several hours.
Posture	Body postures fixed mainly by the flexors.	The tendency is to extension; *arc de cercle;* peculiar attitudes; opisthotonos.
Pupils	Dilated almost without exception and immobile.	Mobile and active.
Tongue	Frequently bitten.	Very exceptionally injured.
Frothing at mouth	Nearly always present, especially after *grand mal* attacks.	Rarely present.
Micturition	Involuntary flow of urine common.	Never.
Defecation	Quite common, but less so than urination.	Never.

	Epilepsy.	*Hysteria.*
Talking	No vocal sound of any kind escapes the patient during an attack of classical epilepsy, save the epileptic cry that is variously modified.	Occasionally a confused medley of words, groans, or somewhat irrational conversation is indulged in.
Necessity for restraint ..	To prevent the patient from suffering bodily injury while he is unconscious.	To control more or less purposeful violence
Temperature	Increased in the majority of cases from half a degree up to 2.5° F. or more immediately after the fit.	Never increased.
General condition on termination	Hebetude, headache, extreme fatigue, muscular soreness.	Ending more prompt. Patient suffers little discomfort.
Convulsive accidents and post-convulsive states	Occasional hemorrhagic extravasations about the face and neck. Scars from injuries or recent wounds. Mental confusion. Automatism. Reflexes, superficial and deep, diminished. Drug eruptions common. The *facies epileptica* is well defined in some chronic cases. Post-epileptic paralysis in some cases.	No hemorrhagic extravasations; reflexes not impaired; mental poise regained at once; no eruption due to drugs. The *facies epileptica* never present. Wounds never received during the attacks. Post-epileptic paralysis never present.

The foregoing diagnostic points apply on the one side more particularly to the convulsions of major hysteria. The points of distinction between the convulsive phenomena of epilepsy and those of hysteria lie wholly along two lines: psychologic and motor. In hysteria, psychologic disturbances predominate in range though not in intensity; in epilepsy motor disturbances, while usually less diversified in character, are more pronounced in degree.

We must not fail, however, to recognize the extremely complex nature of the phenomena that hysterical subjects present, for classic manifestations of this disease are not constant, and especially is this true of the hysterias complicated with epilepsy.

Our greatest difficulty will be experienced in diagnosticating the convulsions of hysteria major from true epileptic convulsions, so that we need scarcely consider hysteria in any other form, unless we should be occasionally perplexed in differentiating the motor disturbances common to both and which are especially

likely to occur in motor epileptics whose disease is of long standing.

Féré has shown that both centripetal and centrifugal transmission in hysterical subjects are retarded, which causes a *slackening* of voluntary movements, necessitating a longer time for the performance of required movements. The same is true of some cases of chronic epilepsy, especially when the disease is largely motor in character. The voluntary movements of hysterical subjects are also marked by *indecision* and *lack of direction*. Both of these likewise apply to the movements of chronic motor epileptics, and in them become even more extreme than in hysteria.

There is another analogous condition essentially dependent upon the same causes operative in the above. Movements of all kinds are *simplified* in character, both in hysteria and in epilepsy, as shown by the fact that neither are competent to perform complex acts which necessitate the execution of several different movements at the same time. Janet has sought to show that hysterical subjects can consciously perform but one movement at a time; a limitation that also applies to chronic epileptics generally during the inter-paroxysmal period.

All these conditions, namely, slackening of voluntary movements, indecision, and lack of direction, and the ability to perform any but simplified movements, indicate the ravages of degenerative diseases, and while possessing great scientific interest, are of but little value when we come to distinguish one convulsive disease from the other.

Alcoholism.—Simple intoxication uncomplicated by convulsions could be mistaken for epilepsy only during the coma period which is similar in some respects in the two conditions. The fact that the patient in alcoholic coma can usually be aroused to some extent and the presence of the characteristic alcohol breath are

usually sufficient to exclude the profound state of
coma that follows immediately upon an epileptic fit.

In severe cases of alcoholism in which there happens
to be a fracture of the skull with deeper coma, the
diagnosis is not easy. In such cases the physician
may save himself some humiliation by not expressing
a fixed opinion until he has had ample opportunity for
observation.

When epilepsy arises as the result of either acute
or prolonged alcoholic indulgence, it is identical in
every respect to the epilepsies induced by other causes,
even though we may call acute alcoholic convulsions
symptomatic only. In such cases less is to be gained
through diagnostic distinctions than through the study
of etiology, upon which the treatment must largely be
based.

When one or more convulsions follow excessive
drinking, and at no other time, the patient should
understand that they indicate acute poisoning of a
most serious kind, and, unless they are checked, will
sooner or later pass into the essential disease, so far as
ultimate results are concerned.

Not infrequently convulsions due to drink continue
after the drink habit is abandoned; in fact, this is
more often the case than not.

After all, the chief point of distinction between most
alcoholic convulsions and those of essential epilepsy, so
far as the ultimate results are concerned, lies in the
relationship in point of time between the indulgence
and the convulsion. When convulsions originally due
to alcohol begin to appear independent of the intoxi-
cated state, we may no longer regard them as sympto-
matic only, but as indicative of a profounder state of
brain instability.

General Paresis.—The chief point of similarity
between epilepsy and general paresis is found in the
epileptiform convulsions that characterize certain

periods of the latter. Chase * speaks of a woman who was treated for four years with the bromids for epilepsy, and who was later thought to have hysterical convulsions, but she proved finally to be a paretic. An express driver mentioned by Folsom had epileptiform seizures for five years, when he became so forgetful and inattentive that he was discharged. Later he developed paresis.

In rare instances the two diseases may coexist, either antedating the other. Chase mentions three such cases, one observed by Christian, one by Markham, and one by himself. The latter was a woman, twenty-eight years old, whose father and uncle were epileptic, while she herself had suffered from epilepsy from the ninth to her twelfth year. She ran a typical paretic career, dying of paresis within eighteen months. Here there was a period of sixteen years between the cessation of the epilepsy and the development of paresis, and it might be difficult to establish any etiologic relationship between the two.

Several men, ranging in age from forty-seven to fifty-five years, have come under observation at the Colony, in whom the epilepsy was due to alcohol, and all presented quite distinct physical symptoms of paresis for years, though none were classed as paretic. They were under observation from five to eight years.

M. J. Q., a male epileptic, entered the Colony in July, 1902. Age, forty-one years; history of epilepsy for four years. Positive syphilitic infection could not be proved, though it was strongly suspected. The patient had been a steady though mild drinker for years. His family history was not good. In nine months at the Colony he had forty-seven seizures, four of them occurring at night; none were *grand mal;* all were mild, a few being psychic and not lasting more than four or five seconds. Several like these occurred

* "General Paresis," 1902, p. 34.

in the writer's presence. The pupils were dilated at each attack, the dilatation being more marked in the left eye than in the right. Reaction to light in the right eye had been slow for several months. He was depressed most of the time, though occasionally would rouse up with some spirit and declare himself capable of again taking up his work as a life insurance agent. A history of some exaltation at the beginning of his trouble was obtained.

His paretic symptoms showed most in his speech, writing, and in general muscular tremors. The latter were especially marked about the lips, face, and hands. He also showed at times a marked facial paresis that appeared independent of his attacks. The specimens of his writing at the beginning of his trouble and four years later, and here presented, show a very pronounced difference.

Several alienists who examined him in June, 1903, twelve months after he came to the Colony, pronounced him a paretic, running a tardy course. It was difficult to differentiate some of his epileptic attacks from the epileptiform attacks of paresis. As a rule, the former were more general, the latter local in their expression, and they did not produce automatism like true epilepsy.

Another point in the differential diagnosis between the two diseases is that the epileptic irritability of temper with strong impulses to violence differs radically from the placid, good-natured demeanor of the paretic while he is passing through the stage of his disease during which epileptiform phenomena are most likely to appear. In epilepsy, too, the inter-paroxysmal period is usually free from evidences of disease, while in paresis this is not the case. Paretic symptoms are continuous.

Fibrillary tremors about the mouth, face, tongue, and fingers in epilepsy adhere closely to the seizure period, generally appearing just before the fit and subsiding completely after it. In general paresis disturbances of motility persist continuously, and steadily grow more pronounced.

Plate 18.

A case of paresis, with right facial paralysis, in which the convulsions so closely simulated those of true epilepsy as to be mistaken for them for a year, when other paretic symptoms became more pronounced. Convulsions first appeared during the thirty-eighth year, and were alternately grand mal and psychic, the latter greatly predominating.

Some motor epileptics in time acquire the drawling, hesitating speech common to paretics, but they show nothing else corroborative of the more fatal disorder. Above all, the epileptic never exhibits the expansive ideas that so often distinguish the mental enfeeblement of the paretic.

Paresis also rarely occurs before adult life, few cases arising under eighteen or twenty, while epilepsy is essentially a disease of early life, more than three-fourths of all cases occurring before the twentieth year.

Notwithstanding the various points of differentiation, we may occasionally meet with cases in which the best aid to diagnosis is time, a study of the patient's antecedents being in the meanwhile of the greatest value.

Syncope.—Ordinary epileptic convulsions and syncopal attacks are not likely to be mistaken one for the other. There may be some confusion, however, in making a distinction between syncopal attacks that cause more or less prolonged loss of consciousness, and some minor attacks of epilepsy that do not involve the motor side. The distinction of these from fainting attacks rests first on the absence of obvious exciting influences. Syncope is most often witnessed in feeble persons and under conditions of mental emotion, overexertion, heated rooms, during exhausting diseases, diarrhea, severe abdominal pains, and heart disease.

The "faints" of epilepsy occur without warning, in the strong as well as in the feeble; they come on at all times and under all circumstances. They do not require an exciting cause, and more often do not disturb body posture, not even causing the eyes to close, a phenomenon never witnessed in simple syncope. In the latter the pulse is apt to be feeble and often fails completely just before the faint. In epilepsy it may be slightly lowered in frequency, though the volume remains good.

Either condition may be preceded by pallor of the face, while slight flushing, which is never present in syncope, occurs at the beginning of some epileptic "faints." The loss of consciousness in this form of epilepsy is sudden and complete, while some confusion and headache follow its restoration. In syncope the loss of consciousness may be preceded by nausea and a feeling of faintness. Nor is there a distinct aura before simple faints, such as often occurs in connection with the special senses in epilepsy. Such aura take a wide range and include many of the forms heretofore described under psychic and *petit mal* epilepsy.

Micturition, which occurs frequently in epilepsy, is unknown in syncope. Automatic acts after the latter are never noticed, while they are more often present than absent after the former. One of the simplest and most common of such acts in this state is that of undressing, the patient often removing part of his clothing before he becomes conscious of what he is about.

In addition to these distinguishing symptoms, the epileptic often shows other severe manifestations of his disease.

Toxemic Convulsions.—I have previously discussed under etiology the convulsions due to alcohol, lead, and certain blood states associated with kidney disease. All of these may cause convulsions epileptoid in character and stop at that, or they may be continued into the establishment of genuine epilepsy (so far, at least, as clinical manifestations and ultimate results go) after the apparent removal of the primary cause. Witness the forms of epilepsy that follow scarlatinal nephritis, those that follow the use of alcohol years after its consumption has stopped, and those that follow a single poisoning of the system with lead (as in the case cited in the chapter on Etiology). It is only when these three causes are active in the pro-

duction of the first few convulsions that our attention in differential diagnosis is required.

When alcohol is the cause, it is generally sufficiently obvious; the convulsions appear after alcoholic excesses, and possess nothing to distinguish them from ordinary epilepsy, save the relationship between cause and effect.

Uremic convulsions are generally accompanied by other signs of the blood state present, while the diagnosis can be made sure of by a thorough examination of the heart and kidneys. In most of these cases the pulse is tense and full, the heart hypertrophied.

Convulsions due to saturnine intoxication are not difficult of detection. The accompanying physical signs, such as wrist drop, colicky pains, the blue line on the gums, and the patient's occupation, are generally sufficient to establish their character. At first they are regarded as symptomatic only; later, like other toxemic convulsions, they may result in established epilepsy.

"It is important to remember," says Gowers, "that convulsions from lead and alcohol may persist after the original cause has ceased to operate, and, in such cases, the effect of that cause and of the repeated convulsions has apparently been to excite in the brain a pathologic state similar to that which exists in simple epilepsy."

Vertigo.—Occasionally difficulty may be experienced in diagnosing aural or auditory vertigo, when severe in form (Ménière's disease), from lighter forms of epilepsy.

As a rule, aural vertigo is associated with labyrinthine disease of a pronounced form, tinnitus, and nerve deafness, which may be slight or severe, of recent origin or long standing. Disturbances, such as ringing in the ears, in such cases are constant and are not confined to the seizure period as in epilepsy.

As a rule, the onset of aural vertigo is sudden, but

it may be slow in passing away, whereas in epilepsy the symptoms disappear at once. A patient may suffer from both aural vertigo and epilepsy (Gowers). In some cases in which there is considerable brain instability and the origin of the disease labyrinthine, the attacks may so closely simulate those of true epilepsy, even to the loss of consciousness, as to create much confusion.

Tetanic convulsions are not apt to be mistaken for epilepsy of the commoner types. They may in very rare instances be confounded with epileptic attacks that are tetanoid in nature, and which have been described with sufficient fullness in Chapter IV under Seizure Types to render further reference to them in this connection unnecessary. As a rule, tetanic convulsions are far more severe in character and of longer duration than those of tetanoid epilepsy.

There is also usually a history of injury in tetanus, such as running a nail in the foot, while the jaws in tetanus remain locked for a much longer time. The history of the case adds points of value in the differential diagnosis of the two affections.

Simulation.—Epileptic convulsions are occasionally simulated by two classes for one of two purposes: either by criminals to escape punishment for crime, or by beggars for personal gain. The former simulate attacks at opportune moments when observers may be led to believe that they are victims of disease; the latter feign epileptic attacks in a crowd, or in selected places where they are most apt to excite the sympathy of those they would deceive.

It is exceedingly difficult to practise simulation in a manner to deceive, for any length of time, one trained in the observance of epileptic phenomena.

The first point in favor of the genuineness of the attack is the previous existence of the disease. If this can be proved conclusively, it is always difficult to say that any subsequent attack is not epileptic.

The first point in favor of simulation is the presence of a motive. Given this, it is always well to question the nature of the attack.

Due attention to the following points of distinction will generally enable us to say whether the attack is spurious or genuine. The simulator chooses the time and place for an attack; the epileptic does not. The simulator seldom falls in a manner to suffer injury, while the epileptic is frequently injured, often very severely. I have previously called attention to the frequency with which scars are met with in the epileptic. These may be found to a limited extent in impostors, but they are rare and seldom if ever met with about the face, which is the favorite site for them in the true disease.

In simulated attacks the pupils are not dilated; the urine and feces not voided; the tongue seldom bitten; the face and neck never covered with petechial discolorations; the epileptic cry is absent; the sensitiveness of the cornea is not impaired; and there is never any degree of post-epileptic or exhaustion paralysis, or any increase in body-temperature. The simulator, in closing his fist during the supposed attack, leaves the thumb outside the fingers, and if the fingers are forcibly opened he will contract them again. In the epileptic hand, the thumb is across the palm and held down by the overlapping fingers. In epilepsy the pupils are nearly always dilated, and immobile to light; urine and feces are frequently voided; the tongue is very frequently bitten; the face and neck in some cases are covered with a petechial eruption; the epileptic cry is often heard; the corneal reflex is generally abolished, and there is not infrequently some marked result of the fit, such as paralysis, automatism, mental disturbance, and aphasia, and in more than half the cases there is an elevation in body-temperature, ranging from .5 to 2.5° F.

The simulator may cause his face to flush or become cyanosed through fixation of the chest muscles, and he can produce a bloody froth by biting the tongue and placing a piece of soap in his mouth; but he cannot cause the facial pallor which is witnessed before almost every epileptic attack, in which flushing is extremely rare.

Another valuable point of differentiation lies in the tone and degree of muscular contractions. The simulator can reproduce tonic better than clonic contractions. By firmly grasping the bare forearms of the patient during the clonic period of contraction, we will find that in true epilepsy the contractions are short, sharp, regular, electric-like, and powerful in degree, as compared with those produced at will. Here they are irregular, mild in degree, and wholly lacking in the sharpness and force that characterize the former.

Heller has suggested that during a suspicious attack firm pressure be exercised on some group of muscles, such as those of the thigh. After a time they will relax, and if an attempt is then made to flex the limb the simulator will contract the muscles again.

In simulated attacks, as in hysteria, pressure over the supraorbital nerve will cause the patient to show distress, while in the epileptic it produces no result.

Hughlings-Jackson first pointed out the loss of knee-jerk and ankle-clonus after severe epileptic attacks,—symptoms which cannot be feigned.

MacDonald reports in the case of Clegg, who was arrested for crime and known as the "dummy chucker," an instance in which skilled observation detected simulated convulsions so nearly like the true disease that they long deceived numerous physicians and prison officials. (See Hamilton's "System of Legal Medicine," Vol. II, p. 404.)

With our rapidly advancing knowledge of epilepsy,

it becomes more difficult for impostors to success-
fully simulate the true disease, while the cultivation
and careful training of professional beggars and crimi-
nals to practise simulation grows constantly less.

CHAPTER XI.

PROGNOSIS.

Recovery Under Treatment. Spontaneous Recovery. Improvement. Favorable Indications. The Prognostic Value of Aura. The Danger to Life. Influence on Longevity.

THE prognosis in epilepsy is more difficult to decide than any other disease, insanity not excepted. It involves not only the question of curability, but the forecasting of numerous other conditions the patient may attain. These differ in different types of the disease and in different individuals of the same type; so that the question of prognosis covers the following: (*a*) recovery under treatment; (*b*) spontaneous recovery; (*c*) arrest of attacks; (*d*) improvement; (*e*) the probable effects of the disease on the mind; (*f*) its liability to a fatal termination; (*g*) its influence on longevity.

RECOVERY UNDER TREATMENT.

Hippocrates* wrote concerning the Sacred Disease: "If it attacks little children, the greater number die. . . . . If youths and young adults, recovery may take place; but there is danger of its becoming habitual, and even increasing if not treated by suitable remedies. Such is also the case when it attacks young children. . . . When it attacks people of advanced years, it often proves fatal. . . . When a person has passed the twentieth year of life, the disease is not apt to seize him unless it has become habitual from childhood. When the disease has prevailed for a length of time, it is no longer curable."

* "Sydenham Society's Translation," Vol. II, p. 850 *et seq.*

Ancient Greek and Roman physicians appear to have taken a more generally favorable view of the malady than has been the custom in more recent times.

It is apparent that we owe the gloomy view of the prognosis largely to the French physicians in the earlier decades of the last century.* Later came more favorable reports from Herpin (1852) and Trousseau (1861) in France, and Russell Reynolds (1861) in England.

As far back as 1778–1780, Tissot almost alone stated that he had cured a great number of epileptics, and nearly a century later Russell Reynolds corroborated Tissot's favorable opinion. Both believed that much could be done toward the arrest of the disease.

In the pre-bromid days it is interesting to recall the high percentage of so-called cures which have been recorded. Thus, Hufeland stated that a cure was effected in 5 per cent. of his cases; Russell Reynolds, in 10 per cent.; Trousseau effected a cure in 20 out of 150 cases, 13 per cent.; and Herpin was perhaps justified in his sanguine belief that he effected a cure in 19 out of 38 cases, 50 per cent. (Turner).†

The same writer states that the almost universal administration of the bromids since their introduction in 1857 has in no way modified the variability of the results obtained. Some of the more recent writers give the following proportions of cures:

Nothnagel, from	4 to 5 per cent.	
Laehr	6	"
Ackermann	7.6	"
Dana	5 to 10	"
Wildermuth	8.5	"
Habermas	10.3	"
Alt	12.5	"

* Maissonneuve, 1803; Pinel, 1818; Esquirol, 1838; Valliex, 1851; and others.

† Turner, "A Statistical Inquiry into the Prognosis and Curability of Epilepsy," "Medico-Chirurgical Transactions." London, Vol. LXXXVI, 1903.

"The introduction of bromid of potassium into therapeutics has remarkably improved the prognosis. Absolute recovery persisting ten, fifteen, or twenty years is rare, but amelioration is frequent" (Féré).

"The facts relating to the cure of the disease are extremely meagre. Few cases can be watched sufficiently long to enable a cure to be confidently affirmed. Moreover, the cases in which the best results are obtained, in which no more fits occur, are precisely those that are never heard of again. Such cases are, I am sure, far more common than is imagined" (Gowers).

The fewer apparent cures under the bromid period may be accounted for in a large measure by greater precision in diagnosis. The later writers excluded from their statistics all symptomatic epilepsies, noting cases of the idiopathic disease only. It is well known that the latter are far less curable than the former.

In his admirable monograph on the subject (op. cit.) G. Alden Turner asks: "Is there a cure for epilepsy?" He answers in the affirmative, and his summary and conclusions are so carefully drawn and are of such value that I give them in substance in the following:

Turner studied 366 cases from out-patient records of the National Hospital for the Paralyzed and Epileptic (London), excluding all save those classed as idiopathic and those in which the patients had not been under treatment at least two years.

They were divided into three series according to the manner in which they responded to treatment: arrested, improved, and confirmed. A family history was found more frequently among those classed as confirmed, but an hereditary history was not found to militate against the prospects of arrest or improvement of the disease in any given case.

The age at the onset of the disease and the duration

of the malady was found to influence the prognosis to a great extent.

Improvement or cure is much more likely to be attained during the first five than during the second five years, although some may be arrested after twenty or thirty years.

Epileptics subject to daily or weekly attacks are least likely to be benefited, while those who go longest without a fit are most likely to be cured or improved. Major attacks are more amenable to treatment than minor ones, next coming major and minor combined, then minor alone.

Long remissions induced by successful treatment, or occurring spontaneously, are not unusual, and have a favorable prognostic value, but are not synonyms of cure.

"From the collected statistics a period of arrest for at least nine years has been fixed as the basis upon which a cure of epilepsy may be established. With this definition of a cure, I regard 10.2 per cent. of epileptics as curable" (Turner).

It would be unreasonable to argue that the length of time, which Turner has fixed upon that should elapse before a cure is pronounced, is not long enough. It should satisfy the most skeptical among those who may not believe epilepsy a curable disease.

I can find no statistics in this country comparable with Turner's; one reason being the lack of age of special institutes for this class; another, the difference in the types of cases studied in city dispensaries, such as the National Hospital for the Paralyzed and Epileptic, London, as compared with those sent to institutions.

Out of 1070 patients first admitted to the Craig Colony, 15 only—less than 1½ per cent.—had suffered from epilepsy less than a year; 197, from one to five years; 267, from five to ten years; 405, from ten to

twenty years; 156, from twenty to forty years; while 30 had had the disease forty years or more.

Notwithstanding the chronicity of the affection in these cases, the results following treatment in many of them were such that I have no hesitancy in stating that epilepsy is not infrequently a curable disease irrespective of its duration, the rule being that recent cases are twice as likely to respond to treatment as chronic ones.

In substantiation of this, in part, the following cases of recovery are briefly cited:

Case I.—A boy, aged ten years at onset of epilepsy, which followed an attack of scarlatinal nephritis. Attacks were mostly *petit mal*, occasionally *grand mal*. For sixteen years they averaged from 115 to 125 a month. The patient's mental condition on admission was not robust, due largely to the effects of the excessive use of the bromids. He was under treatment for two years and two months. During the first month he had 115 attacks, during the second 98, during the third 13, during the fourth, none, and during the fifth, one; after which they ceased entirely. Seven years later they had not recurred. All told, he had 50,000 to 60,000 attacks. The cure in this case seems complete.

Case II.—A girl, aged six years at onset of epilepsy, which followed emotional shock due to fright. Attacks at first were all *grand mal*, later changing to *petit mal*, and occurring at the rate of one a day. The disease had existed twenty-seven years when the patient entered the Colony. She was under treatment two years and a half, and five years after her discharge was well in every respect.

Case III.—A boy, aged fourteen years at onset of epilepsy, which was ascribed to heredity and masturbation. The disease had existed two years when the patient came under treatment. All were *grand mal* and occurred at first six months apart, later changing to one a month. Mental condition was unimpaired; a slight vicious tendency was noted at times. He was kept under treatment for two years

and four months, and five years later had experienced no further trouble.

Case IV.—A man, aged fifty-seven years at onset of epilepsy. Assigned cause, senile changes; probable cause, dissolute living. All attacks were *grand mal*, occurring at first once a month, then once in three months. There was no mental impairment. He was under treatment for two years and four months. Five years later he was perfectly well.

Case V.—A boy, aged sixteen years at onset of epilepsy, which was ascribed to congenital defect and dissolute habits. All attacks were *grand mal*, occurring at first weekly, then once a month. Mental condition was not impaired. He was under treatment two years. Five years and two months later the attacks had not recurred.

Case VI.—A girl, aged nineteen years at onset of epilepsy, which was due to emotional shock from fright. The patient was of a sensitive, nervous organization, easily influenced. All attacks were *grand mal*. They occurred on an average four times a year. The disease had existed a year before admission. She was under treatment one year and nine months, and five years later the attacks had not recurred.

Case VII.—A boy, aged eleven years at onset of epilepsy, which was due to heredity and masturbation. All attacks were *grand mal*, occurring at first weekly, later twice a week. Mental condition was unimpaired. Duration of epilepsy on admission, four years; duration of treatment, one year and five months. Four years later the attacks had not recurred.

Case VIII.—A young man, aged twenty years at onset of epilepsy, which was due to alcohol. All attacks were *grand mal*, severe in character, and occurred two weeks apart. Duration of epilepsy on admission, ten years; duration of treatment, three years. A year and six months later the attacks had not recurred.

Case IX.—A girl, aged seven years at onset of epilepsy, which was ascribed to congenital defect and irritation incident to the second dentition. Only one attack at that time, the second occurring three years

later, after which they appeared every month. All attacks were *grand mal*. Duration of the disease on admission, seven years; duration of treatment, two years. Mental condition was not impaired. Three years later the attacks had not recurred.

Case X.—A boy, aged eight years at onset of epilepsy, which was ascribed to congenital defect and "over-excitement." Attacks were both *grand mal* and *petit mal*, the latter predominating. They occurred at the rate of one a week. Mental condition, imbecile. Duration of epilepsy on admission, twelve years. Three years later attacks had not recurred. Mental condition was improved.

Case XI.—A boy, aged seven years at onset of epilepsy, which was ascribed to heredity and the "stress" attending the second dentition period. All attacks were *grand mal*, occurring at first once a week; later from three to four days apart. Mental condition was not impaired. Duration of epilepsy on admission, seven years; duration of treatment, three years. One year and three months later attacks had not recurred.

Case XII.—A girl, aged eight years at onset of epilepsy, which was ascribed to indigestion. Mother suffered from disease of the spine; father had meningitis at the age of twenty-two. Both parents were nervous and subject to headaches. All attacks were *grand mal*, occurring at first once a year; during the three years prior to admission, from three to five times a year. No mental impairment. Duration of epilepsy on admission, seven years; duration of treatment, three years and a half. Two years later attacks had not recurred.

Case XIII.—A young man, aged eighteen years at onset of epilepsy, which was ascribed to sexual excesses and masturbation. All attacks were *petit mal*, occurring at first from six to twelve months apart; later several times a day. Mental condition was unimpaired. Duration of disease on admission, two years; duration of treatment, two years. Sixteen months later the disease had not recurred.

Case XIV.—A boy, aged fifteen years at onset of epilepsy, which followed an attack of typhoid fever.

Attacks divided between *grand mal* and *petit mal*, and occurring from four to five months apart. Mental condition was not impaired. Duration of epilepsy on admission, five years; duration of treatment, two years. Eighteen months later the attacks had not recurred.

Case XV.—A boy, aged five years at onset of epilepsy, which was ascribed to congenital defect. All attacks were *grand mal*, occurring once a month. Mental condition was unimpaired. Duration of epilepsy on admission, two years; duration of treatment, twelve months. The patient was sent home on trial. Four years later the attacks had not recurred.

Case XVI.—A girl, aged fifteen years at onset of epilepsy, which was ascribed to heredity and the stress of the first menstrual epoch; she was an imbecile, with deficient memory, and all mental processes slow and imperfect. All attacks were *grand mal*, occurring on an average once a month. Duration of epilepsy on admission, two years; duration of treatment, two years and a half. Five years later the attacks had not recurred.

In addition to the above sixteen patients regarded as cured, there were under my observation on October 1, 1903, fifteen others who had gone eighteen months or longer without an attack. All these bid fair to make a complete recovery.

Many are incurable on admission. If we accept the mental condition as a guide in prognosis, fully half of all admitted to institutions like the Craig Colony must be considered incurable. Fully 50 per cent. show mental impairment, being either demented, feeble-minded, imbecile, idiotic, or insane.

Taking the 1286 cases admitted up to October, 1902, and deducting half as palpably incurable, we have 643 more or less chronic cases left. Of this number sixteen patients are reported as cured, and fifteen others bid fair to attain the same end, having gone eighteen months or longer without an attack. Add these and we have thirty-one—practically 5 per cent. of the total number of possibly curable cases.

I have previously stated that less than $1\frac{1}{2}$ per cent. of the total number of patients admitted to the Colony had had epilepsy less than a year when they came under treatment. It seems fair, on the whole, to say that even with an almost entirely chronic class we may expect a cure in about 5 per cent. If all could come under treatment early enough, this percentage could undoubtedly be doubled or trebled.

The Influence of Sex.—Sex apparently plays little part in the prognosis of epilepsy. In Turner's 366 cases, the number of arrests in the men was twenty-six, in the women twenty. At the same time, the number of confirmed cases was greater in the men than in the women.

In the sixteen Colony cases reported as cured, eleven were men; while in the fifteen mentioned as having no attacks for eighteen months, twelve were men. This difference in ratio is explained in part by the greater number of men under treatment. If there is a difference in the curability of the two sexes, I incline to think that it favors the male side. Attention was called in Chapter III to the fact that the disease is more common among men than among women, in the ratio of twenty of the former to sixteen of the latter.

The Influence of Age.—The average age at the onset of the disease in the sixteen cases of recovery was fifteen years. Epilepsy beginning under ten years is least favorable as regards cure or improvement, while cases in which the arrest occurs between the fifteenth and twentieth year are most favorable. From the twentieth to the thirtieth or thirty-fifth year there is a steady decline in the curable cases.*

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* "Those cases of epilepsy that come on before puberty may undergo a change, but those which come on after twenty-five years of age for the most part terminate in death." Hippocrates' "Aphorisms," Section 5, Vol. II; "Sydenham Society's Translation," Vol. II, p. 738.

After the thirty-fifth year, the number of intractable cases diminishes noticeably. This is probably due to not infrequent unessential forms of the disease after this age. "Senile epilepsy is essentially a tractable disorder" (Turner). In this statement I agree.

The following table shows the relation of age, at the onset of the epilepsy and its duration, to the mental condition in 1334 cases: 806 males, 528 females.

Mental State.	Number of Cases.	Male.	Female.	Average Age at Onset.	Average Duration of Epilepsy.
Good	241	142	99	14.1 years.	9.8 years.
Fair	402	251	151	13 "	11.12 "
Feeble	488	282	206	9.7 "	13.1 "
Demented	90	62	28	26.6 "	15.2 "
Imbeciles.......	97	58	39	5.3 "	11.57 "
Idiots	16	11	5	3.76 "	9.35 "
Total	1334	806	528	12.4	11.9

By it we see that the average age at the onset of the epilepsy in those whose mental state was good, was 14.1 years. It also shows that (with the exception of the idiotic class) the disease had existed in the class indicated as "good" for a less time than in any other, the average being 9.8 years. It is an interesting fact to note that those having epileptic dementia acquired epilepsy at the average age of 26.6 years, nearly double the age found in any other class.

The percentage of male and female epileptics in each mental class is as follows:

Good	Male, 17.6 per cent.	Female, 16.8 per cent.
Fair.................	" 31.1 "	" 28.5 "
Feeble..............	" 34.9 "	" 39. "
Demented	" 7.6 "	" 5.2 "
Imbeciles	" 7.1 "	" 7.4 "
Idiots	" 1.3 "	" .9 "

This indicates a greater tendency for male epileptics to retain better mental states under "good," "fair," and "feeble" than female epileptics, while a slightly greater

number of male epileptics reach dementia, imbecility, and idiocy.   This confirms the seemingly greater number of cures among males than among females, for the mental condition has much to do with it.

An additional fact of great interest in this connection is found in a study of the proportion of cases having a neuropathic heredity in each of the mental states given.

Good. Per cent.	Fair. Per cent.	Feeble. Per cent.	Demented. Per cent.	Imbeciles. Per cent.	Idiots. Per cent.
33⅓	37.5	27.4	21.8	24.6	33⅓

This shows that the most amenable cases to treatment—those included under good and fair mental states—most often have a neuropathic heredity, which is confirmatory of the statements of Gowers, Turner, and others.

The Influence of Frequency of Attacks.—The following table shows the relation of mental condition at the time of admission to frequency of attacks.

Interval between Attacks.	Good.	Fair.	Feeble.	De-mented.	Imbe-cile.	Idiot.
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Less than three weeks	58.8	68.6	76.6	77.5	89.2	72
Three to four weeks	22.8	24.4	13.8	15.5	8	16⅔
Over one month	18.3	11	9.3	7	2.6	11.1

It is clear that the more often the attacks occur the less favorable is the mental condition.   Beginning with a percentage of 58.8 per cent. of those marked good and in whom attacks occurred less than three weeks apart, the number runs steadily up as the mental state falls, until we find 89.2 per cent. of the imbeciles having seizures less than three weeks apart. It is also noted that those marked good have the highest percentage (18.3 per cent.) of attacks which appear over a month apart.

The character and time of seizures apparently

modify the prognosis to some extent. It is generally known that *grand mal* seizures are more amenable to treatment than *petit mal* or psychic attacks.

It is also well known that, as a rule, major attacks do not tend so frequently to impair or destroy the mind as do the lesser attacks. Next to *grand mal* in this respect come *grand mal* and *petit mal* combined, while psychic attacks are the least tractable of all. There are some exceptions to this.

For six years a girl, who was eight years of age when the attacks began, averaged from forty to sixty psychic seizures daily, when they began to decrease, appearing four or five times only in twenty-four hours, again disappearing completely for days at a time. Notwithstanding all these, she developed naturally, mentally and physically, grew strong and robust, and at the age of fourteen showed every prospect of becoming rid of the seizures entirely.

In other cases in which the attacks are almost identical with this, mental enfeeblement begins to show in a year or so, and in three or four years the patient is demented.

Why there is this radical difference in outcome between cases almost symptomatically identical, is not known. The personal equation represented in individual stamina, together with the cause, may explain it in part.

Attacks that occur during the day, including those of the early morning (the post-dormitium period), give a larger percentage of cures than those occurring only during the night. I would explain this on the ground that nocturnal attacks, commonest, as we have seen, about three o'clock in the morning, overtake the individual when vitality is at its lowest ebb. The very robust are generally not so apt to have seizures at night.

The Influence of the Catamenia.—The establishment

of the menstrual epoch appears to exert but little influence on the disease, beyond increasing in some cases the number of attacks at that time. It has been quite generally held that the onset of the menstrual function exerts a favorable influence on the disease, but there is nothing within my knowledge to justify such a belief. The opposite is more often true. The table below shows how this period augments the attacks.

FREQUENCY OF ATTACKS IN 1374 CASES.

	Number having Attacks.	Approximate Per Cent.
Attacks weekly or more often	775	57
" every two weeks............	173	13
" " three weeks	61	4
" " **four weeks**..........	**244**	**18**
" " eight weeks	42	3
" " twelve weeks	49	4
" six months or over	30	2

Here the four-weeks' period is greater than any other save the first, in which the attacks occurred weekly or more often, and is due to the influence of the catamenia. It is not uncommon for the menstrual period to be irregular in its manifestations if epilepsy develops during puberty.

The Influence of Marriage.—The marriage of epileptics is sometimes urged for its supposed favorable influence on the disease, but so far as my observation goes, there is nothing to warrant commendation of the practice,—marriage having no beneficial effect on the disease in any of numerous similar cases that I have personal knowledge of. In some of them it seemed to lessen the frequency and severity of the attacks for a short time.

Irrespective of this, marriage confers a license for the creation of a diseased progeny generally lower in mental, moral, and physical stamina than their ante-

cedents. This fact alone should be sufficient to deny the epileptic the right of marriage in fully ninety-five out of every hundred cases in which it is sought.

The value of the aura in prognosis in certain cases is considerable. It is of great value in cases in which motor manifestations are most pronounced, and, as a general rule, the greater the length of time between the appearance of the aura and the fit, the more readily will the case yield to treatment.

In many cases of reflex epilepsy the relationship between discoverable cause and effect is often unmistakable. We need only refer to the list of causes active in such cases to see that this is so. These include gastro-intestinal irritations, urethral stricture, helminthes of various kinds, nasal growths, old cicatrices, recent trauma to the head, etc.

A woman of middle age (to whom reference has before been made) never menstruated. Shortly after the beginning of the period of menstrual life she began to have convulsions described as epileptic, the prodromal periods of which consisted in headache, marked irritability of temper, and malaise, all of which steadily grew in intensity for some days before the catamenia should appear.

She was found to have an impervious uterus, the removal of which, with both ovaries, checked the attacks at once, which four years later had not recurred.

The aura in this case measured the degree of poisoning due to the systemic absorption of the menstrual discharge.

Spontaneous Recovery.—Instances of this kind have been reported, but they are far too infrequent and too uncertain on which to base any hope in a given case.

The tendency of the disease is always toward self-perpetuation,—a fact attested to by the common recognition of habit epilepsy in which the disease

appears after the original cause has been removed, as well as by the teachings of the most experienced neurologists.

Not infrequently convulsive attacks in infancy, which continue to occur after the removal of the exciting cause, disappear spontaneously. Later on, on the other hand, attacks in childhood may suddenly cease to recur at puberty. This is one of the most remarkable features of the disease. In the chapter on the Drug Treatment reference is made to the influence of intercurrent affections on the course of the disease.

The Danger to Life.—The danger to life from epilepsy, according to Gowers, is not great; while Féré says epilepsy is always a serious affection. These two statements are in a measure harmonious. If we hold that a disease which destroys life suddenly and without warning through a single, brief attack, unaided by an accident to the patient at the moment, such as suffocation, or fracture of the skull from falling, and does so in from 3 to 4 per cent. of all who suffer from it, we may consider its danger to life in the same light that Gowers does, for this rate of mortality for a single disease is not excessively high.

If, on the other hand, we feel that epilepsy in any form may suddenly produce death in any case, and that its presence leads indirectly to a fatal termination through its tendency to the establishment of other grave disorders, we may agree better with Féré, who says it is a serious affection.

Epilepsy should never be counted among the diseases that do not threaten life. The most robust of its victims may succumb through a single attack. Several cases which prove this have come under my notice.

It is not safe to argue that the mildness of the attacks always indicates comparative freedom from danger, though, as a rule, accidents in such cases are rare.

A very common cause of death in epilepsy is pulmonary tuberculosis, this being the cause in 24 per cent. of 150 deaths at Sonyea. This would seem to show that epileptics are more prone to diseases of the lungs than those not epileptic. On comparing this ratio—one death in four in epilepsy due to tuberculosis—with the ratio of deaths from all causes in New York State, during the years 1900 and 1901, we find that the percentage of deaths from tuberculosis was about 9½. These figures are not presented as conclusive, but merely as showing a tendency seemingly worthy of further study.

Next to tuberculosis in point of cause, stands status epilepticus. The onset of this is a danger signal likely to be displayed in every epileptic's life. No form of the disease may escape it. It may follow hundreds of violent seizures, or may follow two or three exceedingly mild ones, constituting in the latter cases *masked status*, a condition fully as fatal as status epilepticus of the classical type.

Serial attacks seldom have a fatal termination, though they may pass into status and produce death in that way.

Organic diseases of the heart also seem to play a conspicuous part in causing death in epilepsy. It was found in some form in nearly 10 per cent. of the Colony cases, though it was not the actual cause of death in this number. When death occurs during an attack in an individual who has organic heart disease, it may be difficult to know how much influence to attribute to the epilepsy and how much to the heart disease, though, as a rule, symptoms of asphyxiation usually make epilepsy as a cause comparatively clear.

The manner in which accidents are received by epileptics varies widely, including falling downstairs, from windows, into water, being struck by railway

trains, choking while eating, falling in dangerous positions, and others too numerous to mention.

A boy was standing with his back to the top of a short flight of steps that led to a cellar with a cement floor. A convulsion seized him, threw him down and over the stairs, so that his head struck posteriorly on the stone floor, death resulting in half an hour from a fracture of the skull, which, beginning at the base, almost completely encircled it in a rising line as it extended forward on either side.

Another male epileptic fell downstairs during a seizure and broke his neck.

A man entered a small toilet-room, closed the door, had a seizure and fell in such a way as to brace his feet against one wall and his head against the door, the head being wedged forward and crowding the chin against the chest and right shoulder. When removed he was limp and unconscious and died shortly after as the result of a fracture of the sixth cervical vertebra, as shown by the autopsy.

An adult male epileptic sat on a chair near an iron bedstead that had a right angle rail down the side. He had a convulsion and fell forward in such a manner as to strike with his throat squarely across the sharp edge of the iron rail. When found later he was dead, his entire weight having been supported by his throat against the rail, his legs being out almost at full length.

I have already mentioned the woman who died in an upright position. These cases are cited to show some of the more unusual causes and manner in which epilepsy destroys life. We need not be surprised to find an epileptic dead in any posture, or under any circumstances.

"The question of sudden death," says Brouardel,* "in the course of an attack of epilepsy is of great

* "Death and Sudden Death," 1902, p. 185.

interest. In such cases death is due either to rupture of the heart, or to passage of food from the mouth or stomach into the trachea or bronchi, or to asphyxiation resulting from the position occupied by the patient during the fit, such as may cause the mouth to be pressed into the pillow.

"But besides these, there is another mode of death which, in all probability, sometimes occurs during a fit of epilepsy, namely, sudden paralysis of the respiratory center. Its occurrence was firmly believed in and taught by the late Dr. Hilton Fagge, and it is, perhaps, the best explanation that can be given of the death of those chronic epileptics who are occasionally found to have died in their sleep without any evidence of their having been asphyxiated."

In addition to deaths occurring during sleep, as described by Brouardel, from respiratory failure, I am firmly of the opinion that they may occur in the waking state as the result of the same cause, for such cases have come under my observation, which could not be explained in any other way.

Out of every one hundred epileptics who die, about four do so as the result of a single seizure; about twenty-four as the result of status epilepticus; about twenty-four as the result of some disease of the lungs, chiefly tuberculosis; about twelve as the result of some accident, including suffocation in bed; about ten as the result of some organic disease of the heart; and about twenty-six from all other causes.*

The Influence of Epilepsy on Longevity.—Epilepsy tends to shorten life. It does this in two ways: first, by its effects alone in many cases, as we have already seen; and, secondly, through its establishment so early in life that the majority of those who die through its effects do so before middle age is reached. In 150

* Spratling, "The Causes and Manner of Death in Epilepsy," "Medical News," January 28, 1902.

cases in which death occurred, the average age at death was 29.46 years.

While this is not a sufficient number on which to base conclusive opinions, it is large enough to show that epilepsy tends to shorten life very materially.

The average age at death in epilepsy is noticeably lower than that of insanity. In a State hospital for the insane * in which 134 deaths occurred in 1902, the average age at death was fifty-four years. In a total of 20,086 deaths in the New York State Hospitals for the Insane during fourteen years, 1330 only were under thirty years of age, the average age at death being nearer fifty years than thirty years. The average age at the onset of insanity is between thirty and forty years, while in epilepsy it is between ten and twenty years.

* New Jersey State Hospital at Morris Plains: "Report for 1902."

CHAPTER XII.

PATHOLOGY OF EPILEPSY.

By Thomas P. Prout, M.D., and L. Pierce Clark, M.D.

Introduction. Gross Pathology. Microscopic Pathology. Pathogenesis. Clinical Interpretation of the Present Pathologic Status of Epilepsy.

INTRODUCTION.

It is not necessary to review the history of the pathology of epilepsy here. Most of the theories of the past are now purely of historic interest and the scope of this work will not admit of their general revival. The theories which have formed the real stepping-stones in the progress of our knowledge will not, however, be omitted.

The work of the past has been progressive at every stage; but that of the last few years has seemed to advance more rapidly than that of earlier periods, and we shall have occasion to mention some of the men whose work has played an important rôle in this progress.

The experimental pathologist and physiologist deserve great credit for much of our present knowledge. In judging of their work, however, we must not forget its limitations. Convulsions experimentally produced on the lower animals are not epilepsy, and are unaccompanied by many of its cardinal symptoms. Such phenomena remain simple convulsive phenomena from start to finish, and can no more be regarded as true or idiopathic epilepsy than many other cerebral disorders accompanied by convulsions. These facts

were overlooked by many of the earlier investigators. Brown-Séquard's classic experiments in producing epileptiform attacks in guinea-pigs were open to this charge. The nervous organization of frogs and guinea-pigs is so far removed from that of man that an experimental analogy is hardly to be considered seriously. Such experiments were useful in demonstrating that fits of an epileptiform nature follow certain forms of irritation, and these earlier experiments, of which Westphal's were also a type, form the foundation for much of our present conception of epilepsy. The early belief that epilepsy was a disease of medullary origin, supported as it was by Marshall Hall, Schroeder van der Kolk, and others, rapidly gained many adherents and reached its climax in the promulgation of an epilepto-genetic center in the medulla by Nothnagel. This theory was entangled with many others which are now almost forgotten, such as the opinion that epilepsy was due to a spasm of the arterioles, a diseased condition of the basal ganglia, sympathetic system, or sclerosis of the cornu ammonis.

The discovery of the electrical irritability of certain portions of the cerebral cortex, particularly by Ferrier, Munk, and Fritsch and Hitzig, accompanied and followed as it was by the important clinical observations of Hughlings Jackson on partial epilepsy, and the work of a multitude of other clinical and experimental observers, swept aside all other theories and placed the region of nervous discharge in epilepsy in the cerebral cortex.

In the light of our present conception, therefore, the pathology of epilepsy will be considered under the following heads:

    I. Gross Pathology.
   II. Microscopic Pathology.
   III. Pathogenesis.

IV. Clinical Interpretation of the Present Pathologic Status of Epilepsy.*

GROSS PATHOLOGY.

The range of gross anatomical states found in epilepsy includes almost every possible lesion of the cerebrospinal axis, its membranes and their bony covering. The chief of these are the conditions of atrophy and mal-development following infantile cerebral palsy; localized hemorrhage, thrombosis, embolism, and trauma; localized or general sclerosis, including sclerosis of the cornu ammonis; brain tumor, cyst or skull deformity, either congenital or acquired.

For the most part the gross lesions occurring in epilepsy occupy a secondary place in its causation. Some are more remotely secondary than others; as, for example, the skull changes, the degree of which seems to depend largely upon the age of the patient and the duration and severity of the epilepsy. Other gross lesions, such as those following infantile cerebral palsy and usually involving considerable portions of the cerebral substance itself, do not occupy so remotely a secondary place in the pathology of the disease. Since epilepsy is now regarded as a cortical disease, we can consider only those conditions (gross or microscopic) which involve the cortex, of primary importance pathologically. Therefore the vast majority of gross lesions are but contributory factors in the production of epilepsy, and are of pathologic importance only in proportion to their tendency to produce an unstable nervous organism. The various signs of mal-development are only of importance as evidence of the existence of an unstable and especially sus-

* While we have endeavored to cover the whole field of the pathology of epilepsy, the details of method and technique, more especially touching that portion of this chapter embodying our own original work, will not be considered. For this information the student is referred to the original articles.

ceptible nervous system. Such, for example, are the microscopic anomalies of the cerebral cortex, described by Rancoroni in the brains of criminals and epileptics, also the gross anomalies of brain development embodied in the frequently described abnormalities of configuration, or the gross physical conditions commonly embodied in the stigmata of degeneration. The observations of Ohlmacher on the persistent thymus and the lymphatic diathesis in epileptics undoubtedly belong among the latter, although they may have a somewhat different relationship to the pathology and pathogenesis of epilepsy than the commonly recognized stigmata of degeneration.

Having defined the position of gross lesions in general in their relationship to epilepsy, we may pass to their more particular consideration.

Skull and Meninges.—One of the most constant conditions found in epilepsy is a more or less decided alteration in the texture of the skull, and this is often accompanied by changes in the meninges. The skull is usually thickened, frequently to a great extent, and more especially in the occipital portion, although a general thickening of the calvarium is not infrequent. In some instances the thickening is limited to certain small portions of the skull, causing these areas to present the appearance of an exostosis involving the entire thickness of the skull generally, or the external or internal table especially. In most instances, however, the thickening is general. Binswanger regards these changes as an osteosclerosis.

The density of the skull is usually increased with the thickening, although this does not necessarily follow. This factor depends more particularly upon the age of the patient. Some young epileptics present a markedly thickened skull which is at the same time light and porous. The density, however, appears to be much increased after the twenty-fifth year, and to

be very marked after the fortieth year. The increase in density is accompanied by an increase in the weight of the skull, the calvarium of the epileptic being much heavier than that of the normal individual of the same age.

It is quite probable that the skull changes here described are a sequence to the frequent cranial traumata in the fits and the extensive venous congestion accompanying the convulsion. Congestion of the skull and meninges as a consequence of the epileptic seizure is very constant and most marked following death from status epilepticus or from serial fits. The degree of engorgement depends somewhat upon the age of the patient and the consequent density of the skull. In the younger patients dying during a status period, the vascular engorgement is often extreme, while in those of middle life and old age it is much less.

Deformities of the skull are very frequent in epilepsy, especially if one includes among them the more marked facial asymmetries. Binswanger, Bourneville and Sollier, and Féré have been most active in calling attention to the frequency of pronounced skull asymmetry in epilepsy. Behrend found a pronounced flattening of the back of the head in epileptics, and Müller, in an examination of the heads of forty-three epileptics, found but four that he considered normal.

There have undoubtedly been included with these cases a goodly number of epilepsies complicated by the infantile cerebral palsies, but nevertheless these figures and the consensus of opinion generally concerning the frequency of skull deformities in epilepsy is very striking. Deformity is undoubtedly most frequent in the epilepsies engrafted upon an infantile cerebral hemiplegia and allied conditions. In these cases there is a more or less well-marked asymmetry of the skull, the deformity being most pronounced on

the side of the lesion (Sachs and Peterson). It some-
times happens that the base of the skull takes part
in the deformity, as occurred in a very marked case
published by us (Plate 19, Fig. 1), but such deformity
is infrequent, and we believe occurs only in those palsy
cases of extreme degree, and especially those occurring
in the early months of infancy.

These skull changes are almost always accompanied
by some degree of change in the meninges. This is
especially true of those cases of long standing, and
particularly of the infantile cerebral hemiplegia types,
in which condition the degree of change in the dura
and the pia as well, is quite proportioned to the severity
of the brain lesion and depends much upon its primary
cause. In these cases the meningeal lesions, while not
entirely limited to the areas of the brain involved, are
nevertheless much more severe over these portions,
the dura being thickened and markedly adherent to
the skull and excessively adherent to the pia. In
infantile cerebral palsy, and in many old epileptics,
the dura is so markedly adherent to the skull that
its separation is a matter of difficulty and requires
much care in its accomplishment; even then pieces
of the dura are often torn away with the calvarium.
In a few cases of this character the morbid process
in the dura mater has undergone calcareous degen-
eration with the formation of calcareous deposits
within the substance of this membrane. In one of
our cases this calcareous formation extended over the
whole of one side of the brain surface. The patient,
forty-one years old, had an old infantile cerebral
palsy, the cerebral lesion being not very pronounced.

The formation of a false hemorrhagic membrane on
the inner surface of the dura is not as frequent in
epileptics in general as among the insane. As regards
epilepsy this condition seems to be limited to old
epileptics who present more or less decided mental

involvement with accompanying vascular lesions. The membrane, in the instances that we have seen, has presented the same general characteristics as found in the insane; in fact, they occurred either in asylum epileptics or in epileptics who could have been certified for commitment. The membrane was thin, being never over one-sixteenth of an inch thick, nonadherent to the dura, and entirely unorganized. The point of its origin could not be determined. That a hemorrhagic membrane of this sort might be produced by the fit seems not improbable when one considers the great age of the patients in which it has been observed. It must be remembered that it is entirely secondary, and is so thin that it could not possibly give rise to intracranial pressure.

The subject of skull injury deserves a passing note, since it frequently initiates the epileptic process, especially when occurring over the motor cortex. The rôle of injuries of this sort in initiating the spasm is undoubted and is of special importance when preceded by hereditary causes. Usually both tables of the skull are involved in the injury, the inner table pressing upon the brain surface and giving rise to its irritation. It occasionally happens, as a result of injury, that the inner table of the skull alone is fractured and depressed.

The pia mater presents frequent changes in epilepsy, but none which are constant and characteristic. Of these the most frequent is a certain amount of edema and congestion of the pia, the latter being a sequence of the vascular engorgement incident to the fit. A certain amount of edema is quite common and is especially pronounced in old epileptics and insane epileptics, and is often associated with more or less brain wasting and incidental increase in the cerebrospinal fluid. Thickening of the pia mater, frequently associated with more or less well-marked adhesions

between the pia and the brain surface, is quite common in epilepsy. This is most marked in those epileptics presenting an infantile cerebral hemiplegia, in which condition the lesion is almost constant, but it exists independently of this condition in about 50 per cent. of epileptics.

Opacity of the pia occurs but infrequently in epilepsy as compared with insanity. The reason for this is hard to explain and would seem to refute the suggestion of Bevan Lewis that opacity of the pia is especially frequent in conditions associated with congestion and chronic hyperemia. Further than this, if "we must infer an inflammatory agency" in the production of the extreme degrees of opacity of the pia, we should certainly be warranted in assuming the absence of such inflammatory agency in epilepsy. One sees but seldom the extreme degrees of pial opacity in epilepsy.

Brain.—The old lesions of an infantile cerebral hemiplegia are among the most frequent found in the brains of epileptics. The percentage of infantile hemiplegias and diplegias that become epileptic is variously stated.

In 140 cases Sachs and Peterson found 62 (44.3 per cent.) epileptic. Included in this number were 24 diplegics, 7 of whom were epileptic (29 per cent.), and 11 paraplegics, 4 of whom were epileptic (36 per cent.). Among the 105 hemiplegics, 51 had epilepsy (50 per cent.). Osler found but 35 in 120 of his cases (26 per cent.); Wallenberg, 66 in 160 cases, while Lovett records 26 severe cases in which but one was non-epileptic (96 per cent.); and Gowers states that two-thirds of all cases of hemiplegic cerebral palsies in infants ultimately became epileptic. Among 264 cases seen at the Vanderbilt Clinic, 87 were epileptic (33 per cent.). We are reasonably safe in stating that fully 40 per cent. of all the cerebral hemiplegics

of infancy ultimately become epileptic. In the pro-
duction of epilepsy, the size of the lesion appears of
little consequence compared with its location. Epi-
lepsy may be engrafted upon a lesion so slight as to
escape notice, except by the most careful clinical
examination; on the other hand, the lesion may be so
extreme that one wonders how the patient survived the
original storm. Fig. 1, Plate 19, presents a lesion of this
type in which the whole left cerebral hemisphere was
involved. The less severe lesions may involve almost
any portion of the cerebral substance, although there
is often a distinct tendency to the involvement of the
central convolutions in both hemiplegics and diplegics.
This has been regarded by some authorities (Freud)
as evidence of the vascular origin of the disease. Con-
cerning the exact nature of the original lesion in this
condition, there is no general agreement. We are
probably dealing with a set of conditions the result
of various lesions, which, if we knew the original
excitant, would appear quite simple. Freud, Delettre,
Charcot, Haubner, and others have reported cases
undoubtedly the result of embolism. Abercrombie
reports a case presenting widespread vascular disease
and thrombosis of the superior longitudinal sinus,
and Gowers suggests thrombosis of the cortical veins
as the underlying cause. According to Collier, recent
cases show no signs of thrombosis. Freud thinks
all the infectious cases are embolic and that the hemi-
plegic cases are thus best explained.

Strümpell's theory of the existence of a truly in-
flammatory state, a polioencephalitis which is an
analogue of the poliomyelitis of children, has received
little corroboration aside from his own school of
workers. Wernicke describes a case associated with
poliomyelitis anterior, but it is a notable fact that no
considerable number of similar cases are on record.
Since the influenza epidemics there have been some

similar cases recorded, chiefly in adults; the final
lesion in these cases, however, is very different from
that of the palsy lesions of childhood (Collier).

The rôle of congenital syphilis in causing these palsy
lesions is very subordinate, if it has any real existence
in fact.   It seems reasonable that if many of these
post-natal cases (hemiplegics) are to be explained by
the occurrence of cerebral hemorrhage, we are also
to assume the action of some deteriorating influence
upon the vessels, such as hereditary syphilis, which
renders them especially susceptible.   Weyhe thinks
some cases are to be thus explained; we are in accord
with Sachs, however, in believing that there is little
or no evidence in corroboration of this view-point.
The record of congenital syphilis among this class of
cases is too slight to warrant us in transferring Weyhe's
autopsy statistics of meningeal hemorrhage in con-
genital syphilis to cases of infantile cerebral palsy in
explanation of the primary lesion.   In our opinion,
it is probable that embolism or thrombosis following
the acute infectious diseases will explain most of the
hemiplegias, while the possibility of an acute infec-
tious process in this condition is not to be lost sight of.
A large proportion, if not all, of the diplegias are con-
genital, resulting from tedious and difficult birth, the
lesion being a hemorrhage due to instrumental de-
livery or to a thrombus formed in the vessels during a
period of suspended animation.

The percentage of infantile cerebral cases among
the epileptics at the Craig Colony is about 8 per cent.
Out of 1872 epileptics seen at the Vanderbilt Clinic,
87 were hemiplegic (4 per cent.).   Of the insane
epileptics at Morris Plains, N. J., 10 per cent. were
hemiplegic.   The proportion of these cases to epilepsy
in general is best represented by the figures from the
Vanderbilt Clinic.   It is a notable fact, in this con-
nection, that the smaller the lesion in infantile cerebral

palsy, the more severe the epilepsy and the greater the liability to its occurrence. This fact, first suggested by Bravais in 1824, has been revived by Sachs and more recently corroborated by Freud. It is worthy of note that in many epileptics of the palsy type the lesion is so slight that no real palsy exists (infantile cerebral palsy without palsy, according to Freud).

Sclerosis of the cornu ammonis is one of the most frequent gross conditions found in epilepsy. It was first described by Mynert and subsequently by Sommers and Bourneville, all of whom laid much stress upon the condition. More latterly Bratz, Wooster, Féré, and Chaslin have investigated the lesion and, while impressed with its importance, are nevertheless inclined to let it gravitate to its proper place. Féré regards it as merely an expression of a widespread sclerotic change throughout the cerebral cortex. The frequency of its occurrence varies considerably, depending on the class of epileptics observed. Bratz reports fourteen instances in thirty-two cases examined. The degree of sclerotic change varies greatly and in many instances the condition is unilateral. In stating our own opinion regarding this lesion, we would amplify the opinion of Féré and say that while sclerosis of the cornu ammonis is but a local expression of a widespread sclerosis of the cortex, nevertheless such sclerosis is of a secondary character. This subject will be further considered under the microscopic section.

Tumor, Cyst, and Vascular Lesions.—Tumors of the brain giving rise to a true epilepsy are comparatively rare. On the other hand, epileptiform manifestation as a result of tumor developing in the motor cortex is not infrequent. The development of true epilepsy as a result of tumor requires that the tumor be located in or very near the motor region, of comparatively

small size, and of slow growth. Such tumors must
necessarily be non-malignant and are far more liable
to development in the meninges than in the brain
substance.

The most frequent forms of brain tumor are sar-
comata or gliomata which may occur primarily and
may develop in the brain substance, or occur second-
arily to malignant growths elsewhere. Carcinomata
and sarcomata frequently develop metastasis in the
brain. The epileptiform manifestations incidental to
such formations, however, do not belong in the domain
of epilepsy.

Cyst formation in the brain cortex of epileptics
deserves special mention. Old epileptics not infre-
quently present small cysts in various portions of the
brain. This seems to occur especially in those epi-
lepsies developing in middle life. They are probably
secondary to vascular changes in the smaller arterioles
of the cortex. The changes described in one of Collin's
cases belong to this class. In senile epilepsy there is
usually an extensive arteriosclerosis, ultimately giving
rise to plugging of the vessels of the cortex.

Following death from a series of severe epileptic
seizures or from status epilepticus, the cerebrospinal
system often presents numerous small punctate hemor-
rhages. Most of these are located in the cortex, but
they may occur in any portion. Weber has recently
called attention to the frequency of these hemorrhages
in status, and has laid special stress on their frequent
location in the bulb. He would not only make these
lesions the direct cause of death, but would also thus
explain the transient paresis (exhaustion-paralysis)
following serial fits and status. We do not believe
this is warranted, since, while we have seen numerous
cases in which this lesion did occur, we have seen
numerous others in which it could neither be demon-
strated macroscopically nor microscopically. Cor-

tical punctate hemorrhages may be a factor in explaining certain slight permanent paralyses following status, nevertheless these phenomena of disturbed motion are, for the most part, to be regarded as exhaustion phenomena, as described in another portion of this work.

Epilepsy as a result of cerebral hemorrhage or embolism in the adult is comparatively rare, but does occur. The lesions produced differ in no particular from those uncomplicated by epilepsy, a surface hemorrhage being more prone to the production of epilepsy than one in the cerebral substance.

MICROSCOPIC PATHOLOGY.

With the beginning of the last decade epileptologists were almost in accord regarding the cortical origin of the epileptic fit, but as to its underlying pathology in the cortex there was great diversity of opinion. The theory of the origin of the fit in a pronounced cerebral gliosis (Féré, Chaslin, Sommer) was accompanied in greater or lesser degree by conceptions of its origin in "fine molecular changes" undemonstrable by the microscope (Binswanger, Gowers, Jackson) and of a more or less evanescent character. The natural successor to this was the autotoxic theory (Voisin, Agostini, Cololian, Krainsky), the advocates of which have ignored cerebral changes and sought to explain epilepsy solely on the basis of an auto-intoxication. This has been accompanied by a movement on the part of experimental physiologists and pathologists (Prus, Hering, Redlich, Bischoff, Rothman, von Bechterew) who have attempted the determination of the nature of the spasm, its exact seat of origin and mode of transmission. We may ask ourselves at the outset what these various groups of workers have accomplished.

The first group of observers has demonstrated the

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extreme frequency of cortical gliosis in epilepsy. They have not demonstrated its universal occurrence, but we shall have occasion to note further on that there is some reason for assuming its unexceptional occurrence in cases of any considerable duration and severity. Some of these observers (Alzheimer, Bleuler) have also demonstrated the great frequency of cortical cell changes, such as chromatolysis, in almost all of their cases; however, there has also been a tendency to emphasize the rôle of gliosis in epilepsy, and there has been much theorizing on the possibility of the glial overgrowth being the essential underlying factor.

The second group of observers has recognized the insufficiency of the findings and theories of the first group, and has attempted to get nearer to a real explanation of the facts by the enunciation of another theory launched partially in the realm of dynamics. They have demonstrated nothing regarding this theory and its relationship to the pathology of epilepsy.

The third group of observers has recognized also the insufficiency of the known microscopic findings in explaining the pathology of epilepsy, and has entered the field of chemistry and toxicology in search of an explanation. They have demonstrated the heightened toxicity of certain secretions and excretions of the epileptic, and have attempted, without success, the demonstration of the exact substance entering into the intoxication. That the toxicity of certain of the body fluids is greatly heightened in epilepsy, seems well established, but the exact rôle of these toxic substances in producing the epileptic fit has not as yet been proved. It is to be noted that this work is still in its preliminary stage, and that experimenters have not progressed beyond the demonstration of the toxicity of certain of the body fluids. The work of this group of observers will be further considered under pathogenesis.

The fourth group of observers has done much to push forward our knowledge of the essential nature of epilepsy. Epilepsy may be due. to a toxin or auto-toxin, but the manner of its action must remain a mystery, unless we can know something of the nature of the fit. The portions of the cortex and the cell elements involved, the nature of the fit, and the tracts over which it is transmitted—all these have been problems which these workers have approached with more or less success. The bearing of their work upon the microscopic pathology of epilepsy will warrant us in presenting some portions of it more in detail.

Prus has recently undertaken the demonstration of the paths over which the fit generalizes, with a view to determining the rôle of the sensory and motor elements in the production of the spasm. In order to do this he severed the pyramidal tracts on both sides at various levels from the internal capsule to the spinal cord; notwithstanding this, however, the development and generalization of the fit were not interfered with on electrically irritating the cortex. From this he concluded that the fit in epilepsy did not generalize by means of the pyramidal tracts. This was further supported by the following experiment: After cutting through everything in the peduncles except pyramidal and pontine fibers, he found that he was not able to produce a fit on electric irritation of the cortex. If the motor cortex was irritated the corresponding muscle groups contracted, but no epileptiform manifestation followed. From this he concluded that neither the pyramidal tracts, the fronto-bridge tracts, nor the temporo-occipito-bridge tracts take any part in the transmission of the impulse in epilepsy; that the impulse is transmitted by means of tracts which lie entirely without these, and that these tracts course in the dorsal portion of the peduncles.

Hering, and more recently Bischoff, have each cor-

roborated in large measure this work of Prus. The
work of Starlinger, who demonstrated the non-exist-
ence of paralysis in dogs in which the pyramidal tracts
were destroyed, was in a measure anticipatory to
that of Prus, in proving the existence of tracts other
than the pyramidal tracts capable of transmitting
motor impulses. Recently the more exact identity
of these tracts has been proved by Redlich, Rothman,*
and others, and the original findings of Prus have in the
main been established. Having demonstrated to his
own satisfaction that the fit generalized over other than
the ordinary motor paths, Prus undertook the demon-
stration of the nature of the fit; i. e., whether irrita-
tion of the sensory or of the motor elements of the
cortex induced the epileptic spasm. In order to
elucidate this question, he painted the cortex with
various agents which impair the sensibility of the
sensory elements. While cocain was applied locally
no fit could be produced on electric irritation of the
cortex, but the individual muscle groups in the periph-
ery would still respond on stimulating corresponding
portions of the cortex. Bromids diminished the irrit-
ability of the cortex. He concludes that the starting
point of the fit in epilepsy is either in the end tufts
of sensory nerves in the cortex or in the sensory cells
of the cortex and not in the motor cells or fibers. This
fact is corroborated by clinical evidence furnished by
the decided influence of the bromids upon epilepsy in
general (explainable by way of its effect upon the sen-
sory elements of the cortex), and by the fact that the
initial manifestation of epilepsy is often an aura which
always belongs in the sensory realm.

The preceding facts are very suggestive that epi-
leptic convulsions are a manifestation of a diseased
state of the sensory elements of the cortex.

* The fasciculus-sulco-marginalis descendens and the fasciculus-rubro-
spinalis (v. Monakow) correspond to the "extra-pyramidal tracts" of Prus.

Plate 19.
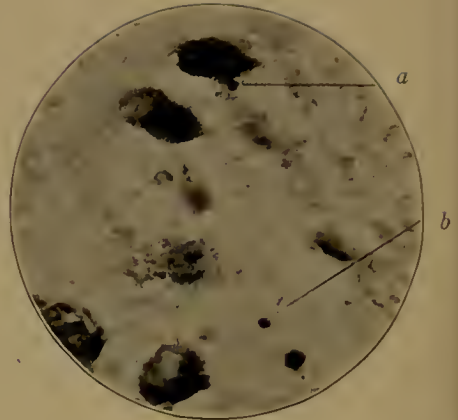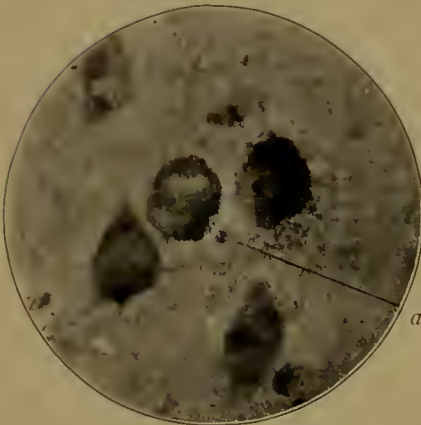
Fig. 1.

Fig. 2.

Fig. 3.
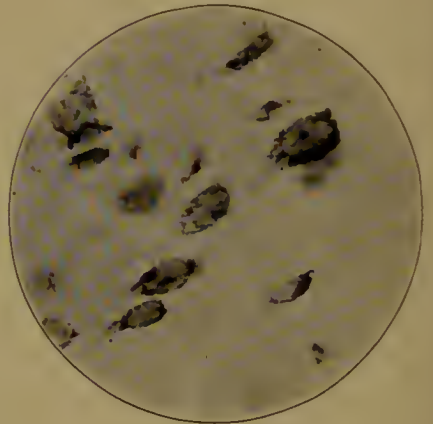
Fig. 4.

Fig. 1.—The lesion of a severe case of infantile cerebral hemiplegia involving the whole left cerebral hemisphere; the right cerebellar hemisphere is secondarily atrophied.

Fig. 2.—Cells lying in the outermost portion of the second cortical layer (case of status), showing two abstracted nucleoli (*a* and *b*). Cells swollen and granular; the nucleus and its limitations poorly defined.

Fig. 3.—Group of cells lying in the lower portion of the second cortical layer (case of status), showing cells greatly swollen and granular with abstracted nucleolus (*a*) lying without.

Fig. 4.—Group of normal nerve cells, second cortical layer. Section made from a case of accidental shooting in which the patient died instantly.

Nissl stains.   Photomicrographs $\times$ 1000.

A demonstration of the particular sensory elements involved would strengthen this hypothesis. We are confronted, then, by the question: What particular elements of the cortex are involved in epilepsy, and are such elements, broadly speaking, sensory or motor in character? The results of our study of the cortex of twenty epileptics from the Craig Colony, with this question in mind, warrant the following statements:

The most striking changes presented by the cortex of the epileptic are found in the cells of the second cortical layer, cells distinctly sensory in type. In patients dying during a period of status epilepticus these changes are most decided. The cells are swollen in many instances to twice their normal size, the nucleus being especially large and granular, with indistinct outline. The limitations of the nucleus are often difficult to determine, the chromatic substance has almost disappeared from the body of the cell, and this portion of the cell appears ragged and poorly outlined. The most striking changes are found in the nucleus. In addition to being granular, swollen, and poorly outlined, the nucleolus is often absent, having been abstracted from the nucleus in the process of section-making (Plate 19, Figs. 2 and 3). This occurs in status cases two or three hundred times more frequently than in sections of normal brain (Plate 19, Fig. 4).

The significance of these changes is obvious. The most important alteration is found in the nucleus of the cell of the second cortical layer and other cells of that type. The change affects the intranuclear network, destroying it and rendering the nucleolus a loose body within the nucleus, so that it is readily abstracted from it in making the section. The ultimate disappearance from the cortex of cells so seriously altered is to be inferred. The particular involvement of the nucleus in this process is a fact of the first importance. Biologic facts teach us that "the

formative power of the cell centers in the nucleus"
and that it is therefore "to be regarded as the especial
organ of inheritance"; that "it plays an essential
rôle in chemical synthesis"; that "digestion and
absorption of food and secretion cease with its removal
from the cytoplasm" and that "fragments of proto-
plasm deprived of the nucleus die" (Wilson, Ver-
worn). When, therefore, morbid processes attack
the nucleus, the vital center of the cell, the portion
essential to its life is threatened; if this continues for
a period the cell is ultimately destroyed. We are
justified in assuming, therefore, that the essential
poison in epilepsy is a nuclear poison, which shows a
special predilection for certain delicately constituted
cells of the cerebral cortex, typical of the second layer.

Many authors have described chromatolysis fol-
lowing the epileptic seizure. It is always present
following serial fits and is especially marked in status
cases, in which condition it is often so decided that
the large Betz cells of the motor cortex appear like
denuded cell remnants, the body of the cell being
greatly shrunken. Changes of this character are always
general.

In the light of recent investigations (Marinesco,
Lugaro, Ewing) we are to regard these changes as
nutritional in character, brought about chiefly by the
suspension of function on the part of the nucleus which,
as we have previously mentioned, presides over the
processes of nutrition and assimilation within the cell.

An extensive invasion of the cortex with leukocytes
is a very frequent condition, especially following status
and serial fits. This was especially marked in some
of our own cases, leukocytes being often found in the
pericellular and perivascular lymph spaces and through-
out the cortical substance. It is highly probable that the
phagocytic property of the leukocyte is here called into
play to remove the products of tissue metamorphosis.

A neuroglia hyperplasia is a very frequent microscopic change in epilepsy, and was especially marked in some of our cases. It is usually manifested by a very pronounced glial overgrowth, especially in the outer cortical layer. The outer layer often appears of unusual depth and the neuroglia fibers dip down deeply into the cortex from cells lying in the outer layer. There is often a subcortical glial overgrowth, especially pronounced in some imbecile epileptics, in whom a pronounced general gliosis is common.

In the light of what has gone before, this glial overgrowth is easily explained. The nerve cell once destroyed is never replaced. The vacancy caused by its disappearance must be taken by cells capable of proliferation. It has been experimentally proved that the neuroglia will proliferate in response to chemical irritation. The great increase in the neuroglia in epilepsy occurs, therefore, in all probability, in response to (a) the action of toxic elements peculiar to epilepsy upon the neuroglia cell direct, and (b) as a direct sequence of nerve cell destruction. With improved methods neuroglia proliferation will probably be demonstrated in every case of epilepsy of any considerable duration and severity.

The question of the phagocytic function of the neuroglia is suggested by some recent work of Mallory. He has shown that many cells of endothelial type and origin become markedly phagocytic in response to the action of certain toxic substances. This theory seems possible, especially since the suggestion of W. Ford Robertson of two distinct types of cells in the neuroglia—one of epiblastic and the other of mesoblastic origin. He also points out that the mesoblastic cell is phagocytic. It would seem possible, therefore, that we may have cells now considered as belonging to the neuroglia that become phagocytic under provocation.

The conclusion seems warranted that epilepsy is a disease-state of the sensory elements of the cortex, and that the impulses constituting the discharge phenomena are peculiar to such disease-states and are transmitted over other than the ordinary motor paths. The elements of the cortex most seriously involved are certain sensory cells of the second cortical layer, some of which are destroyed during the epileptic process.

PATHOGENESIS.

The presence of a predisposition is not sufficient to account for epilepsy, hence the necessity of invoking some agent which excites the organic cellular anomaly of the cortex to morbid activity. Even in traumatic cases it is not necessary to discard this explanation. Indeed, in such cases a faulty chemotaxis of the cortical cells themselves probably constitutes the inciting factor. From modern data, our present view-point that the pathogenic agent rests in the domain of chemical pathology is logical, but it must point the way more definitely before further progress is possible.

We are permitted therefore to make only the broadest generalizations as to the inciting agent in epilepsy. Although the chemical studies are but tentative, they are suggestive. The blood, investigated by Ceni, Herter, Donath, Cololian; the urine by Agostini, Voisin, Peron, Mairet and Vires, K. von Quonge, and Saike; the gastric contents by Bond, Agostini, Rachford, Putnam and Pfaff; the sweat by Cobitts and Movrojainnis, and finally the secretions of the ductless glands, particularly the thymus, by Ohlmacher and many others, have been and are still under investigation.

In general terms it may be said that the majority of good observers in chemical pathology believe that blood, sweat, urine, and gastric contents are hypotoxic

in the inter-paroxysmal state of epilepsy; hypertoxic just before the fit, at the time of and for a variable period just after the fit. There is no agreement, however, upon the agent or agents producing this alteration in toxicity. Some believe it due to carbamate of ammonium (Krainsky, Weber), others find cholin and neurin which they believe to play a rôle in the causation (Donath); but the presence of the two latter agents is probably due to the same cerebral disintegration in epilepsy as in general paresis and senile dementia, where they are present in great excess (Mott, Halliburton).

Ceni claims to have demonstrated an autocytotoxin and an anti-autocytotoxin in the epileptic blood which does not exist in a free state in the plasma, but is latent chiefly in the blood-cells from which it is set free at the convulsive period by a ferment.

Xanthin and paraxanthin have been found in epilepsy as well in the migraines (Rachford), but other investigators while finding it in some cases do not find it in others and deny that it plays an important rôle in the etiology of either disease (Putnam and Pfaff). The disease has been attributed to a leukomain poisoning (Bond and Agostini), to acetone (Deutsch, von Jaksch, and Futcher), and to a diminished alkalinity of the blood (Lui and Pio Galante).

Extensive clinical work under control and fixed conditions has yet to be done. From the nature of the cortical cell changes we have a right to expect that the inciting agent or agents will be very active nuclear poisons.

CLINICAL INTERPRETATION OF THE NEWER PATHOLOGIC FINDINGS.

The remote relationship of previously discovered gross brain lesions to that of epilepsy has been fully dealt with elsewhere, and we shall concern ourselves

only with correlating clinical data with the newer facts in pathology and particularly with the cortical cell changes already described.

The constancy of the cortical lesion in all the varying states of epilepsy, from the mildest type to the most rapidly fatal status, warrants broad generalization in regard to the nature of epilepsy itself.

That the essential phenomena of the disease are sensory in character, and that the motor manifestation is secondary, has now the support (1) of the logical hypothesis of Hughlings Jackson in which the second layer cell has inhibitory relationship to the motor cell, and also acts as a promoter and regulator to its activity. The physiologico-pathologic work upon animals by Prus and others, and (2) the pathologic evidence in the histo-cytologic examination of the cortex of epileptics at the Craig Colony. The disease may be considered therefore a highly organized sensory motor reflex of the cerebral cortex.

The sedative action of bromids, experimental and clinical, furnishes therapeutic proof that the afferent side of this reflex-arc is operated upon either peripherally, or, what seems more probable, upon the sensory type of cells in the cortex itself. Bromids control the fits by reducing the intensity of afferent impulses to motor cells; the latter are without doubt immediately responsible for the muscular convulsion in the fit. Furthermore, when the bromids produce acute or chronic poisoning the state produced is quite comparable to the immediate and remote effects of the disease itself, and as a consequence they have been charged with the mental stigma of the disease (progressive dementia). The bromids acting upon the sensory elements aid cerebral inhibition. The fact that they are of little avail in brain diseases more or less definitely motor in character, such as myoclonus, paralysis agitans, and the post-hemiplegic disorders of

motility, is corroborative of their primary physiologic action upon the sensory cortical cells.

The anatomic evidence of the persistence of the large pyramids of the cortex which are largely motor in function, is proof of the autonomy of these elements. They may and often do undergo marked shrinkage in epilepsy, but are quickly restored to their former inter-paroxysmal stage in the rest period. As they never disappear from the cortex in the same degree as those of the sensory type, paralyses other than that of the transitory exhaustion type (and another form to be considered) are of rare occurrence.

The evidence that epilepsy is a diffuse lesion of the entire cortex is of practical moment and aids much in explaining the complex and bizarre symptoms, and also the inutility of narrow principles in treatment. It is not unlikely that further research may indicate certain areas of the cortex which are most diseased in special cases, particularly those portions of the cortex most highly specialized in sensory and sensori-psychic functions. There is as yet, however, no adequate pathologic evidence for this.

Clinical facts constantly urge that attention be directed to the sensory elements. An order of muscular march in the fits shows only the successive order of discharge in the motor centers; it usually remains identically the same for years, yet we know that the disease, if unrelieved, steadily undergoes important modifications, shown in a continuous destruction of cortical elements. Turning from the motor symptoms to those of the sensory we find that the aura, the loss of consciousness, and the degree and character of mental changes, are the sensory factors considered in diagnosis and prognosis. Some physio-psychologic means of recording the latter would aid much in estimating the degree and character of cortical destruction. The failure of surgical interference, even

when focal symptoms still remain as shown elsewhere in this work, is explained largely because the cortical changes are diffuse, and in other areas and in other cortical elements than those classed more especially as motor in function. A coincidence, however, of damage to both sensory and motor elements in the Rolandic area in traumatic epilepsy explains largely the good results in surgical interference in acute cases where the predisposition is at a minimum.

The most serious clinical phase of the cortical pathology is due to the ultimate disappearance of the cortical cells; their destruction explains many of the permanent symptoms of the disease, especially the slowness, awkwardness, and incoördination of muscle movements, and the progressive mental failure (dementia) which is seen in so many epileptics. The disorder of motility in chronic cases amounts to a *paralysis* in many instances. The local and general exhaustion present after local or general fits (especially seen in those parts which participate most in the convulsion), are true exhaustion-paralyses in type, congeners of paralyses from destructive lesions of motor cells and tracts, but the sluggish, awkward, and incoördinate movements present in chronic epilepsy are really consequent upon cell destruction in the second layer, which leaves the motor elements indefinitely informed of the sensations to which they should respond. The damage or loss of sensory cells not only permits cortical motor cell overaction, as seen in the fit, but also leaves these motor elements uninformed of the normal nature and character of movements required. Analogous theoretic explanations were first urged for the ataxia of tabetics by Jackson. The anatomico-pathologic difference is that in ataxia the destruction is anterior in point of time in the sensorimotor reflex-arc. The dementia of epilepsy is analogous and is usually commensurate with the defective motility; it

has been erroneously ascribed as the cause of the latter rather than an incident and coincident of it.

Finally, the modern pathology of epilepsy gives sufficient ground for the present broad but empirical treatment of the disease, which consists largely in overcoming hereditary tendencies as far as possible, and excluding toxic and autoxic agents and administering a carefully detailed plan of living plus such degrees of sedation as may be necessary in individual cases. In the light of the pathogenesis (although as yet it but foreshadows its fruition) and the histo-pathologic changes already known, with their certain results in impairment of normal cerebral functions if not checked, the importance of the most comprehensive and early treatment is obvious.

The missing links in our knowledge of epilepsy are its pathogenetic agents and the organic anomaly of the cortex which constitutes its predisposition; these two factors still hold the mystery of frequent relapses. The gap between its terminal gliosis and the toxic and autoxic agents has been narrowed by recent knowledge of the initial cortical cell changes, which particularly concern the nucleus.

We are warranted in drawing the following conclusions regarding the pathology of epilepsy:

1. Epilepsy is a cerebral disease attended and followed by profound and diffuse cortical degeneration.

2. The morbid changes concern chiefly the destruction of the nuclei of the cells of the sensory type from which the primary departure of the disease originates. Its terminal pathology is a progressive gliosis more or less marked and diffuse.

3. Epilepsy is essentially a sensory phenomenon with a motor manifestation.

4. Its etio-pathology rests with a variety of toxic or autotoxic agents not as yet definitely isolated or determined.

5. The disease is engrafted upon a cortical organic cellular anomaly which is induced largely by a faulty heredity, the exact anatomic nature of which is not known.

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CHAPTER XIII.

THE GENERAL TREATMENT OF EPILEPSY.

Control of Patient of First Importance. Regulation of the Patient's Habits and Diet. The Value of Proper Exercise. The Purpose and Scope of Medico-pedagogic Treatment. The Young Epileptic's Especial Demand for Education and Training. The Mental, Moral, and Physical Failure of the Epileptic without Care.

THE treatment of epilepsy lies along three distinctive lines: *General*, *Medical*, and *Surgical*, and we will consider them in the order named.

In speaking of the general treatment of epilepsy, we refer more particularly to the treatment of the epileptic himself than to the treatment of his disease. The importance of the former has been steadily increasing since the epileptic first came under especial care in this country in institutions designed for his needs, twelve or fifteen years ago. Up to that time the medical treatment of the disease was held to be of paramount importance, since, indeed, it was the only one then known to be at the physician's command. But now, while drugs play a conspicuous and valuable part, they are regarded by the best among those whose familiarity with epilepsy is greatest, as often falling short of fully meeting the requirements in all cases, so that the aid of other agencies must be evoked. These will be described in detail in the present chapter, for their value is clearly beyond dispute, while the knowledge of their use, variously modified as they must be to meet changing types of cases, is not nearly so universal as it ought to be.

To be sure, the physician in general practice is often handicapped by his inability to control the

patient in the manner desired, as is fully possible only in special institutions; but this apparent disadvantage is not nearly so great as it seems at first glance, for, as a rule, the general practitioner has a more intelligent class to deal with than the institution physician. To the latter, patients are often sent only after their control has largely been lost at home.

Barring this single difference, the valuable features of treatment now so greatly in vogue in colonies of different kinds are fully as valuable to the epileptic treated at home as to the epileptic whose constant atmosphere is some form of colony life.

The Control of the Patient most Essential.—We may start with the following proposition, applicable without exception to every case alike: *The more absolutely the physician is permitted to control the patient in every respect, the more promising the hope of amelioration or cure.*

This may be accomplished in one of three ways, depending largely upon the age, general intelligence, and character of the patient. It may be accomplished first by securing the aid of the patient himself, having him fully understand what he must do in order to get well, and at the same time show a perfect willingness in literally carrying out every direction imposed upon him. Such patients will be quite frequently encountered, though they do not constitute the majority, for the reasons that epilepsy being essentially a disease of early life and so often causing mental impairment in a few years' time, the patient lacks the judgment and discretion of maturer years, and mental faculties unclouded by disease.

But there still remains a very considerable number whose disease does not appear until after puberty, whose general development and education up to that time were good, and whose attacks are of a type to permit the retention of a comparatively sound mind,

good judgment, and an ambition to employ every effort to recover. It is in such cases as these that the physician will receive the patient's full aid, always a factor of prime importance in the attainment of the best results.

In other cases, especially children, and those who bear marked evidences of the effects of their disease, such as mental enfeeblement, impaired will power, deficient judgment, incapacity for reasoning, and moral depravity of various kinds; who have no vocation and little or no education; who have no power of applying themselves for the accomplishment of any definite purpose—these constitute the most difficult class to handle with success, and it can be done only by placing the patient under an environment, even without his full acquiescence, where his habits will be regulated to the uttermost detail.

In many cases the family can give the attention required, though oftentimes, especially if there are other children in the family, the patient is a constant source of solicitude and anxiety. Irrespective of the influence of the invalid child on its immediate associates in other ways not desirable, the danger from assault by the patient is sometimes great, making him a constant and positive menace in the home. This, to be sure, is not always the case, but it often is; and when we recall the essentially explosive nature of the disease, we can understand the risk every home assumes in keeping an epileptic among its members.

Another factor that operates strongly against the advisability of treating some epileptic patients in their homes, is the unavoidable substitution of parental sympathy for the necessary degree of mild, yet wholesome discipline that most epileptics stand so greatly in need of. They do not require this discipline so much because they are inherently headstrong, depraved, or incorrigible, but because their affliction ever

tends to give them a degree of temperamental obliquity of an unhappy kind that comes and goes most noticeably along with the seizure periods, which is often constantly present during the inter-paroxysmal period, and which is unquestionably improved under the influence of self-restraint when such influence is forced through proper disciplinary measures to act.

Teaching the epileptic the power of self-control is one of the most valuable things we can put in his possession. In some epileptics the exercise of inhibition alone may prevent the occurrence of a threatened attack.

While the home care of the epileptic—his supervision by a member of the family—can at times be carried out successfully, by far the most satisfactory plan outside of special institutions is to place him under the care of a nurse or attendant especially trained in the care of such cases. Only under this condition can the patient remain in his own home to the best advantage.

Colony Life.—The final and most satisfactory plan of securing full control of the patient is to place him in a colony designed for all types, except the insane, or in one for selected cases only.

Bethel Colony at Bielefeld in the province of Westphalia, Germany, and the Craig Colony at Sonyea in Livingston County, New York, furnish the best examples of general colonies at this time; while the smaller colony at Chalfont St. Peter, near London, is an example of those for selected cases only. The Craig Colony admits all epileptics except the insane, receiving on this basis from 85 per cent. to 90 per cent. of all who apply. Chalfont St. Peter rejects 60 per cent. as undesirable, either because of their unfavorable mental condition or of their inability to become useful factors in colony life (Turner).

Both types of colonies have particular spheres of

usefulness, the larger and more cosmopolitan of the two being in greater favor under state or governmental supervision; the smaller, best suited to private or semi-private philanthropic ends.

The principles of colony life in either case are the same. It would be interesting in this connection to briefly review the history of the colony movement from its inception in France in 1849 up to this time, but for full information on the subject the reader is referred to the writings mentioned below.*

We outline the purposes and advantages of colonies as follows:

1. They provide home life, simple and elemental in form, for they take the individual from his own home, unsuited to his peculiar needs, to homes especially designed to supply such needs.

2. They tend to preserve individuality, the one thing institutions are most apt to destroy. In colonies, individuals, not units and numbers, are integral, essential parts of the whole.

3. They provide vocations for all who require them— vocations ranging from the simplest to the most complex, from weeding the cabbage-patch to the making of brick and the construction of houses; they provide education that begins at the alphabet and ends in some profession which guarantees self-support; they provide amusements and recreations not bound by rules of necessity, regularity, and formality; they provide for the organization of homes in a way to throw congenial spirits into pleasant companionship, and to place the less fortunate, the less desirable, in a class apart. Ideal colonies provide, in short, for the in-

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* "Bethel Colony at Bielefeld, Germany," by F. Peterson, "Medical Record," 1887. "A Colony of Mercy," by Julie Sutter. "The Care and Treatment of Epileptics," by Hon. William P. Letchworth, LL.D., 1900. "Report of the Proceedings of the Annual Meeting of the National Association for the Study of Epilepsy," 1901. "Institutions for Epileptics," by the author, "Journal Amer. Med. Association," 1902.

finity of simple, daily, homely necessities that go to create and keep cemented together the best features of communal life. In doing this they are more than homes in that they provide the highest treatment for disease, training along all lines, encouragement wherever required, upbuilding of character, and the necessary healthful restraint for all who need it; and they are less than institutions in that they do not destroy the individuality that counts for more than all the rest, but singles it out and bases all help upon its character.

The epileptic, the chronic insane, and the feeble-minded can all be successfully colonized. The system is not one of rigid requirements in little ways; it may suitably be modified to meet the peculiar needs of the three classes named. But in the care of all classes its fundamental features are the same. To modify these would be fatal to the system itself.

The chronic insane and the feeble-minded can be cared for in other ways, in a few buildings, or, what is less desirable, in a single large building; but epileptics cannot be cared for successfully, or even with partial success, in any other way than under the colony plan. For the epileptic it is ideal; for the insane and feeble-minded it would be a long step in advance of methods now in use, and would ultimately prove as valuable as it has for the epileptic.

The site of the colony should be removed a reasonable distance from centers of population. The defectives who live in them have no place in the strenuous life. They are infinitely better off in localities where the hum and roar, the wear and tear and strain put upon the strong in the outside world do not penetrate. They also require seclusion from the things that lead to dissipation,—things that everywhere abound in city life, but find their lowest ebb in rural communities.

The colony estate should be large—fully an acre to each individual.

Colonists should attend schools and acquire trades. At Sonyea they are graded into classes, with teachers for each class. The boys attend the Sloyd School, which fits them in two or three years to take up advanced carpentry. They build houses and make furniture. Others are apprenticed to the painter, the upholsterer, the printer, the blacksmith, the mason, the engineer, the laundryman, and other artisans; all becoming in time proficient in their several fields. They have short hours of labor and are led, not driven, to work. They have plenty of time for amusement and recreation, indulging in baseball, football, indoor games of all kinds, reading, getting up stage plays, and spending their time when free from duty as they desire, or as people in ordinary communities do.

Just here comes in the most valuable part of it all. All are free; that is, all who have enough mind to permit them to exercise self-care to a safe extent. There are no barred windows or locked doors, save for the few whose safety demands it; and yet under this liberal system less than one per cent. leave the colony without permission. This is the finest feature of the entire scheme, the feature of perfect freedom. There is some trouble, to be sure, for we are dealing with human nature diseased, human nature pathologically modified, which makes illogic conduct easy, even necessary, because it is unavoidable. It is my conviction that many institutions would do greater good if they would adopt measures of greater freedom toward those they care for. Witness the benefits derived from the abolition of forms of bodily restraint applied to the insane, and forms of restraint in the way of walls and high fences about hospital buildings for the insane during the past quarter of a century. A notable revolution in this respect has been at work since Pinel struck the manacles from the hands of the insane at the old Salpêtrière more than a hundred

years ago, when he said, "I am convinced that these lunatics are so unmanageable only because they are robbed of air and liberty, and I dare to hope much from the opposite means of treatment."

From a knowledge acquired through experience in an institution where home life was impossible, and from a knowledge of the home life prevailing at the Craig Colony, I have come to believe that the home instinct is the last of the natural desires to die even in a people who suffer mental enfeeblement through chronic and far-reaching diseases.

It may be claimed that the cost of building cottages or small houses is greater than for one building accommodating hundreds, but this opinion is erroneous. It has not been the case at Sonyea.

The model colony, in recognition of the peculiar needs of epileptics, will provide for three classes of dwellings, not including a hospital, as follows:

Class I.—Houses in this class should hold anywhere from twelve to eighteen patients, and these patients should be of the best; good enough in every respect to assume the entire care of the household in all its details, under the general supervision of one nurse or employe, who should look after sick patients and make observations of the seizures for the physicians in charge. These small cottages should be as home-like, cozy, and attractive as possible, and no room should hold more than two persons, while half of the patients should have single rooms. Approximately 20 per cent. of all in the colony will live in houses of this kind.

Class II.—Houses in this class should be large enough to accommodate from twenty-five to thirty-five persons. These dwellings need to be more numerous, for in them the great middle class, numbering from 60 to 70 per cent. of the entire population, will find a home. They should have light and airy rooms, large hat and coat rooms, smoking and sitting

rooms for men, and plenty of closet and shelf space—
for most of these people find comfort in looking after
their individual possessions—ample verandas, reading
and writing rooms. To lessen the danger from fire,
they should not be grouped closer together than from
seventy-five to one hundred feet, nor more than from
100 to 150 feet apart, in order that they may be
heated from one central point, and that all the patients
of a group may use one shower or rain bath, there
being several groups in all. Some houses need indi-
vidual bathtubs for sick or feeble persons; but great
economy of time will be gained if central bathing and
heating plants are established.

Class III.—This class should consist of infirmaries—
buildings combining home and hospital—for the use of
perpetually bed-ridden, paralytic, or other helpless
cases. These houses should be large enough to care
for from 10 per cent. to 20 per cent. of the epileptic
colony, and one such structure should be provided
for each sex. Each building should contain isolation
rooms for cases mentally disturbed, a feature common
to many epileptics.

In addition to homes for patients, colonies should
have the following buildings:

1. A house for administrative work, containing no
patients, to be regulated in size by the ultimate size
of the colony.

2. A hospital building, complete in equipment and
thoroughly modern in every respect, for the treatment
of acute medical and surgical cases.

3. Industrial buildings, including laundry, power
plants, and bakery, shops for new and repair work
in wood, iron, printing, upholstery, leather, plumbing,
painting, electric and steam engineering, tailoring,
shoemaking, and dressmaking.

4. Special industrial buildings for educational pur-
poses; those for men to include Sloyd School, trades

school, carpenter shop, and nearly all the above to be used in an elementary way by apprentices to fit them for higher and more practical work along similar lines. For the women, laundering, sewing, knitting by hand and machine, darning, rug-weaving, basket-making, and the domestic arts should be engaged in. A laboratory for scientific research is an important feature.

This brief exposition of the colony system shows the broad principles under which the epileptic should live. For obvious reasons all cannot take advantage of it. At the same time, there is no reason why the individual patient under the general practitioner's care should not have colony methods pointed out to him in a way that, if persistently followed, will be very sure to materially accrue to his benefit.

The idea of invalidism should not under any plan of treatment be allowed to take root in the epileptic's mind, but should be constantly combatted. He should not be made to feel that he is in a class apart—unlike other persons. Such conceptions will be naturally forced upon him fast enough through his being denied the privileges of the public schools, attendance at church, public exercises of all kinds, and even exclusion from the family table.

His expulsion from the common life augments his morbid temperament, his irritability, his inability to express his thoughts, his moping, his apathy, his natural tendency to a lack of sociability, all of which tend to aid in his often being reared in deplorable ignorance. Neither should he be forced forward in a way that makes him conspicuous and subjects him to unpleasant comment and ridicule. The epileptic child has often to bear the latter from its playmates. Some feel the sting of it, others do not. Those who do should be given protection from it, for it may do lasting injury in cases of sensitive disposition.

The medico-pedagogic treatment of the epileptic is

essential for many reasons, the chief among them being that it is more than educational—it exercises a curative power of well-determined distinction in selected cases.

The first question is that of determining the form of education best suited to the individual case, and the extent to which it should be carried.

As previously stated, the cases of essential epilepsy of any considerable duration are comparatively few in which there is not some mental impairment. This must be the case in any disease that so injuriously affects the brain.

The epileptic is subject to marked variations in his capacity to learn. In any aggregation of epileptic school-children there will almost certainly be some whose powers of comprehension are periodically inactive and sluggish in the extreme, and at times suspended altogether, to be restored to normal activity after the effects of the impending attack that forced the inertia have subsided.

The peculiarity of this alternating state of the perceptive powers shows the necessity not only for individual as distinguished from group or class instruction, but for constant repetition in many cases of the matters taught.

The epileptic must be taught largely under a system of reiteration pursued with the greatest patience. We must be satisfied in many cases with a minimum of result under a maximum of effort. In some cases the seizure appears to completely and instantly destroy the memory of a thing learned but a short while before. This shows in a measure the degree of disorganization that occurs in the central nervous system as the result of the seizure. In cases of epileptic dementia such destruction is carried to an extreme degree, so that in suitable cases all mental power may be effaced permanently and completely in a few years' time. In

other cases in which the attacks are more motor than psychic, the mind may suffer almost no impairment under frequently repeated attacks of many years' duration. The recognition of this point aids us in selecting patients most likely to learn under any system of instruction as distinguished from those who will be least likely to do so, or who, having acquired some knowledge, will be most apt to suffer its loss in a comparatively short while.

The very fact that epilepsy may affect, in greater part at least, a single faculty or part of the body, should be sufficient for making it appear that, in order to base a system of education for the epileptic upon correct physiologic principles, we must plan it along many lines. We cannot single out any particular faculty and cultivate it to the exclusion of others. Education must be neither wholly intellectual nor wholly physical, but a definite combination of the two in a way that enhances the value of each. The necessity for doing this is further augmented when we remember that the type of the disease in certain cases (being such at one time as to warrant the development of certain faculties) may later change so as to affect, perhaps destroy, the very faculties to which we have paid so much attention. As a rule, this is not to be feared, but its possibility must not be forgotten.

Without laying it down as a fact applicable in all cases alike, it is generally better to educate the epileptic in a physical or manual way than in a way purely intellectual.

This statement is based on the carefully observed results from the two systems of education in vogue at the Craig Colony among from 80 to 100 younger epileptics during a period of eight years. Most of these, it should be noted, were partially or wholly dependent, thus requiring a form of education valuable for economic and therapeutic reasons combined, the latter being the main object always in view.

Wholly outside any economic considerations whatever, physical education for all classes of epileptics, no matter what their pecuniary or social status may be, is, for reasons purely physiologic, the best for them in every respect. Manual training directed along proper lines is intellectual also. It confers upon the muscular system a practical knowledge ready for use at a moment's call, and *which is not dissipated or destroyed by a single or by repeated seizures, as often happens when knowledge is intellectual only.*

Take, for instance, the value of the Sloyd system of education * in this respect. Its purpose is complexly educational, as may be seen by the following:

1. It requires the student to make a drawing in detail of the object he aims to produce, whether it is a simple wedge, a penholder, a saw-handle, a cabinet of drawers, or a piece of carving. To do this he must learn to use with precision the delicate instruments needed for the purpose. This cultivates his power of muscular sense and coördination, the very thing the established epileptic requires, since disorders of motility, muscular inefficiency, and inexactness of effort are not uncommon results of the disease.

2. It trains the mind and eye in the habit of attentive observation. Epileptics are especially prone to show a lack of concentration of effort, a very notable difficulty in fixing their attention continuously upon one thing (aprosexia), which makes it necessary to drill them assiduously in order to enable them to carry a given task to its completion.

3. It encourages the faculty of thinking largely through the mathematical requirements of every piece of work executed, because of the complete diagram which must first be made of it, giving its dimensions to a scale.

* As defined by an American authority: "Sloyd is tool-work so arranged and employed as to stimulate and promote vigorous, intelligent self-activity for a purpose which the worker recognizes as good."

4. Finally, when the image of the full requirement is definitely fixed in the student's mind, he must create in wood, metal, plaster, clay, leather, or what not, the object itself; so taking the whole process into consideration we can readily understand that its educational value for the average epileptic is very great.

While it is usually taught such persons only in the schools of special institutions, it can be taken up by the individual patient in his home as well. If he possesses any mechanical faculty whatever, he may successfully carry out the system alone.

The mentally feeble child, to which class many epileptics belong, is especially incapable of comprehending abstractions. All instruction, therefore, must be presented in a concrete form which will enable it not only to see, but, when possible, to grasp in the hand as well as in the mind. Many of the games and occupations of the kindergarten are consequently of service; but while the normal child exercises its own spontaneous activity through these occupations, those who are mentally deficient in an apathetic way have to be stimulated to action by the force of imitation.

Our system of education, then, starts on physiologic lines, first addressing itself to the *culture of the external senses*, then to the *coördination of muscular movement*, and finally to the promotion, through proper exercises, both of the manual and mental activities (Shuttleworth).

The supreme object in these cases is to substitute, if possible, the purposeless, irregular movements with those of a definite purpose. In this way only can the inability to fix the attention be overcome. In undeveloped epileptic children in whom nervous irritability is so great as to give rise to destructive tendencies, the irregular, purposeless, illegitimate expenditure of energy should be turned into construc-

tive channels. Children of this type may be gently coaxed into building something of definite form with blocks for the simple pleasure of destroying it later.

Other simple educational implements and means for the very backward may be employed, such as the peg-board, size-board, form-board, graduated wooden rods and blocks, domino-boards, simple exercises in mathematics, drawing, reading, and writing; also object-lessons that frequently bring under the child's notice all the simple familiar objects of daily life should be employed in suitable cases.*

These elemental measures apply more particularly to the epileptic child whose disease has developed before the seventh or eighth year. More advanced methods are called for in children who have had some school advantages before they became epileptic. In these, the system of instruction is not essentially different from that followed in ordinary schools, though we will find that they fall far short of making the progress of the ordinary child. Epileptic students about the age of from sixteen to twenty generally show the mental development of children of from eight to twelve years.

In a class of 60 girls at the Craig Colony, the average age of those in the first (highest) grade was nineteen years; in the second, sixteen years and a half; in the third, fifteen years and a half. With one or two possible exceptions, none of these could reach the standard of work required of normal children.

Whatever the mental status or age of the epileptic student, the scope of his instruction should be broad

* Reference works on educational training: Shuttleworth, "Mentally Deficient Children," 1900; Bourneville, "Recherches cliniques et théra-peutiques sur l'épilepsie, l'hystérie et l'idiotie," "Report Bicêtre," 1901; Froebel's "Letters on the Kindergarten," 1901; the writings of Sequin, Dean, Anna Snell, Gill, and reports of New York, Massachusetts, Illinois, Pennsylvania, and other State institutions for educating the defective classes.

and should lean to the cultivation of the physical rather than to the purely mental side.

Systematic outdoor life requiring a modicum of activity is better than any form of sedentary occupation and should be required whenever possible. Excessive physical labor that causes exhausting fatigue is no more advisable than severe mental labor which for the epileptic is always bad. Such occupations as gardening, seed-gathering, farming, wood-cutting, brick-making, dairying, herding cows and sheep, ranch life, working about lawns, grading, cultivating flowers, raising chickens, and the like, are all admirable for epileptics who show or can be induced to cultivate fondness for them.

The records at the Craig Colony show that attacks among male patients are noticeably less in summer when outdoor activity is possible to the fullest extent, than in winter when it is not.

They also show that female patients do not experience the same degree of improvement that male patients do because of the sedentary life of the former.

TABLE SHOWING COMPARATIVE FREQUENCY OF ATTACKS
IN DIFFERENT SEASONS OF THE YEAR
IN THE TWO SEXES.

	Male.	Female.	Total.
January	5718	3250	8968
February	5320	2929	8249
March	6363	3258	9621
July	4735	3439	8174
August	4922	3635	8557
September	4723	3427	8150

Indoor occupations that are suitable include printing, painting, shoemaking, glazing, carving, modeling in clay, carpentry in all its branches, upholstering, plumbing, broom-, brush-, and basket-making, rug- and carpet-weaving. Under supervision, selected patients (as at Sonyea) may act as engineers, firemen, masons' assistants, teamsters, machinists, book-

keepers, seamstresses, and they may do all manner of housework, including cooking. Epileptics should enjoy as much recreation as possible. They may safely play tennis, golf, baseball, football, basket-ball, indulge in athletic contests of almost every form, practice in the gymnasium (which is heartily recommended under proper caution), and take part in any of the indoor amusements common to other people.

Emotional girls and young women are better for not attending the drama and other places of amusement that excite the emotions. Attacks may be induced in this way.

Like other defectives, epileptics are fond of music, and its influence on them is beneficial. Stage plays are useful in cultivating self-confidence and improving articulation.

Smoking by the young epileptics should be forbidden. If the habit is not contracted until after adult life and moderately indulged in (cigarettes excluded), it often does no harm. In some cases we have known smoking to bring a calm that the individual stood in need of.

Marriage.—The physician may be called upon to give advice on this point. If so, his position will be difficult. He can best answer by viewing the matter from two points: (1) That of the probable effect it will have on the disease itself when present on either side; (2) the liability of its transmission to the off-spring. As to the first, he may say that, as a rule, no good accrues to the individual, so far as the disease is concerned, from the assumption of the married state; and as to the second, that the marriage of epileptics puts the strongest possible premium upon the creation of epileptic progeny. There are rare cases in which parents contract the disease comparatively late in life, in whom it may not for some time be the essential malady, and whose offspring are quite sure

not to be epileptic. Such instances, however, are rare, and require the most delicate selection under medical investigation.

Assume the proper attachment between man and wife, one being epileptic, and eliminate the possibility of epileptic offspring, there may be good reasons why marriage in such cases should be sanctioned. But who can doubt the extreme rarity of these conditions? On the whole, it is better in every way that epileptics should not marry.

Diet.—The question of diet is one of great importance, though not of the same value in all cases. Improved methods of administering the bromid salts of late years have greatly lessened the need for dietetic restrictions. There is no question that the ill effects of the bromids, as shown in part by the manner in which under injudicious administration they impair digestion, have often led to the assumption that imperfect diet was causing or aggravating the disease, whereas the blame should have fallen upon the drug itself and its power to produce gastro-intestinal disorders.

A few years ago, when it was customary to receive patients into the Colony, to whom large doses of the bromids had been administered for years without regard to the possible obviation of their toxic properties, it was noted that stomach disorders with their attendant results—anemia, headaches, loss of flesh, insomnia, general feebleness, etc.—were far more frequent than later on when the bromids were more rationally used. The diet in most cases had later been corrected also, but even under the same diet improved ways of giving the bromids greatly lessened stomach disorders.

All epileptics do not demand the same detailed dietetic considerations, although the principles are essentially the same in all. To say that no epileptic should eat meat would be as wrong as to say that all

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should eat it. I have been unable to determine that different foods have any specific effect on epilepsy itself beyond that which they have on the organism in general, the state of which influences the disease favorably or otherwise.

The epileptic first of all demands a sound, vigorous body, perfect in all its parts. His food requirements are greater than those of almost any other class. He is justly credited with having a voracious appetite. The reason of this is probably found in the necessity for constantly restoring bodily energy that through periodic "nervous discharges" is constantly going to waste. That he should eat much is a demand founded on the results of his disease.

It is essential that he be properly guided in his food habits. Oftentimes more can be done for him in this than in any other way, especially in cases in which the disease is autotoxic in origin and in which dietetic indiscretions bring on a seizure. In many such cases the nutritional balance—the fine line that separates physiologic processes in nutrition from those that are pathologic—is extremely delicate, requiring time and patience and repeated trial to find its adjustment.

Without attempting to review the whole process of digestion, the following practical suggestions from a general hygienic and therapeutic point of view are worthy of consideration.

Assuming first that the patient is free from gastro-intestinal disorders of any kind—constipation, chronic diarrhea or dysentery, indigestion, flatulence, or catarrh—he can best be kept so, or, if he suffers in such ways, be largely or wholly relieved by observing the following to the letter.

1. Regularity in eating is of first importance; irregularity tends to disease.

2. Moderation in the quantity eaten should be strictly enjoined in every case in which there is a

tendency to overeat—a fault common to many sufferers from the disease.

3. Food should never be taken under stress of limited time—every morsel of it should be thoroughly masticated before it is swallowed. Epileptics of inferior grades are prone to bolt their food almost wholly unmasticated.

To formulate a practical dietary for epileptics it must be made easy and inexpensive of preparation, for it will be required daily for years. It must contain a proper proportion of the three groups of food stuffs—albumin, carbohydrates, and fats. While vegetarians (like the Hindoos) live and thrive principally on non-nitrogenous substances, such as vegetables and fruits, other races (like the Esquimos) live almost wholly on nitrogenous articles, but history shows that the highest attainments of the human race have emanated from those who have lived on a mixed diet (Virchow).

LIST OF FOOD ARTICLES SUITABLE FOR EPILEPTICS.

FOR BREAKFAST.

Fruit.—Grapes; grape fruit; oranges; apples, peaches, pears, cooked or raw, as usually served; prunes.

Cereals.—A modicum of cereals only is advised. While they are nutritious, used to excess they tend to weaken the digestive system. They require to be thoroughly cooked, and include oatmeal, cracked wheat, hominy, grits, etc.

Eggs.—These are of especial value and may be eaten in almost any form, except fried,—soft boiled, scrambled, poached on toast, omelet soufflé. According to the manner in which they are prepared, eggs are digested in from an hour and three-

quarters to three hours, hard boiled and omelet soufflé taking the longest time (Penzoldt).

Bread.—Ordinary baker's bread, not too fresh; Graham, gluten, entire wheat, and corn-bread muffins. Toast in any form is especially good.

Liquids.—Coffee, tea, milk, cocoa, chocolate. A minimum of coffee and tea is advised. The best drink is milk. It contains all the elements of a typical diet, including albuminous substances in the form of casein and serum albumin, fats in cream, carbohydrate in the form of lactose or sugar of milk; salts, chiefly of lime, and water. It is easily digested, remaining in the stomach but little longer than plain water, which makes it an ideal food. Coffee and tea should be made quite weak and lightly sweetened.

FOR DINNER (NOON).

Oysters.—These may be raw or cooked any way, except fried.

Soups.—Vegetable, thoroughly cooked; tomato, plain; tomato bisque; consommé; pea; potato; vegetable, corn, and lamb broth. Mock turtle and other rich soups should be avoided.

Meats.—Most epileptics can eat some meat not only with impunity, but with benefit. It should be a part of the noon meal only and consist either of lamb, beef, mutton, fowl of almost any kind, or baked or broiled fish. The quantity of meat should be small in either case. Fowl or game should be broiled, roasted, or fricasseed,

never fried. Rich sauces should be ab-
jured. Simple meat juices do no harm.

Vegetables.—Potatoes white or sweet, spinach, carrots,
parsnips, onions, lima beans, peas, okra,
squash, string beans, asparagus, egg-plant
(broiled), green corn, tomatoes, and tur-
nips. *Avoid cucumbers and cabbage in
any form.* All vegetables should be thor-
oughly cooked.

Relishes.—Olives, radishes, lettuce, celery; peanuts
and almonds, plain or salted.

Dessert.—This should always be simple and consist of
light puddings, such as bread, farina,
rice, tapioca, custard; cheese and crackers.
Pudding sauces should be plain and never
contain wine or alcohol in any form.

FOR SUPPER.

The last meal of the day should be eaten not less
than an hour and a half or two hours before retiring.
It is best for the epileptic to retire on an empty stom-
ach, so this should be the lightest meal of the day,
consisting of toast, crackers, bread and butter, eggs
in any of the forms named for breakfast; prunes,
stewed or baked apples, pears, weak tea, cocoa, choco-
late. Hot corn bread and boiled hot rice two or three
times a week, if desired.

WHAT NOT TO EAT OR DRINK.

Small berries with hard seeds, such as blackberries
and raspberries.
Cake in any form.
Pastry in any form.
Pork, veal, ham, or anything fried in grease.
All alcoholic drinks; even the lightest beer should
be avoided.

The principles underlying a suitable diet for epileptics are simple. They embody the use of bread-stuffs, butter, milk, fruits, cereals, etc., to the exclusion of too much meat, pastry, and other less digestible articles. The above diet is not presented as final for adaptation *in toto* in each case, but under trial it can be made to suit any in which the requirements are not unusual.

CHAPTER XIV.

THE MEDICAL TREATMENT OF EPILEPSY.

Prevention of Attacks.   Treatment of Attacks.   Drugs: The Bromin Preparations.   Their Physiologic Action.   The Prevention of Bromid Intoxication.   The Hypochlorization Method.   How the Bromids Act.   The Opium-bromid Treatment of Flechsig.   Codein.   Borax.   Nitroglycerin. Chloretone.   Zinc.   Sodium Biborate.   Urethane.   Solanum Carolinense. Simulo.   Chloral Hydrate.   Amylene Hydrate.   Belladonna.   The Coaltar Derivatives.   Iron.   Chloroform.   Electricity.   The Relief of Eyestrain.   Serotherapy.   Hydrotherapy.   The Influence of Concurrent Diseases.

THE medical treatment of any disease in which the etiology and pathology are as obscure as they are in epilepsy must be more or less empirical.

As will be seen later on, this applies even to the use of bromid in epilepsy, a drug that has enjoyed well-nigh unusual prestige in the treatment of the disease for more than half a century.

Other forms of treatment not strictly medical, and yet not surgical, are reviewed in this chapter.

The Prevention of an Immediate Attack.—When an aura precedes the attacks a sufficient length of time, the seizure may not infrequently be prevented. One method is by inhalations of nitrite of amyl. For this purpose, nitrite of amyl pearls are carried in the pocket and one is broken in the handkerchief when occasion arises; or the patient keeps with him a small vial with ground-glass stopper containing a piece of cotton saturated with the drug. In the majority of cases, however, the patient is powerless to apply any remedy himself. With the aura there usually comes confusion which destroys intelligent action.

It is assumed that the nitrite of amyl acts beneficially

by flooding the brain with arterial blood, a potent agent for modifying the action of nerve elements (Gowers).

Attacks can sometimes be arrested in other ways. When they begin in one extremity, they may be checked by tying a ligature tightly about the leg or arm involved. It has been advocated for such patients that they wear a double piece of tape looped about the arm above the elbow, the ends hanging down below the sleeve. As soon as the fit is felt coming, the string is pulled tight. Now and then the repeated arrest of fits in this way is said to have a permanent effect.

Faucher recommends hypodermic injections of $\frac{1}{10}$ to $\frac{1}{15}$ of a grain of apomorphin as a speedy means of arresting an attack. I have found it effective in some cases.

Brown-Séquard and Buzzard tried to permanently abort attacks by keeping a blister about the limb.

As a rule, however, little or nothing is gained by checking an approaching attack. It may be advisable at times on account of the patient's environment to do so, but when checked, later attacks seem more severe, thus evidencing a cumulative action.

Several male patients under the writer's care banded together to prevent attacks in one another by rubbing and slapping the side of the face and neck of the victim threatened with the seizure. The plan was not infrequently effective for weeks at a time in some of them, though I cannot say that any permanent benefit came from it.*

> * *Iago.*   My lord is fallen into an epilepsy:
>   This is his second fit; he had one yesterday.
>   *Cas.*   Rub him about the temples:
>   No, forbear;
>   *Iago.*   The lethargy must have his quiet course;
>   If not, he foams at mouth, and by and by,
>   Breaks out to savage madness.
>   "Othello," Act IV.

I have previously called attention ("Albany Medical Annals," Vol. LIII) to the value of an occasional convulsion in certain cases. In some patients the fit acts as a safety valve that unquestionably permits escape from insanity. I referred to the matter in part as follows: "In many cases the convulsion seems to come as the termination of an obscure autotoxic cycle which varies in duration in different individuals and bears some relationship to the ascending period of the *folie circulaire* of the French. It seems that the specific cause of the fit in these cases is something that permeates the entire organism; something that comes and goes; that grows rapidly in intensity, exerting a pernicious influence on the patient by making him act out of harmony with his normal state, until the limit is reached and the mind loses its direction and control. The power of inhibition being finally destroyed, the nervous storm breaks with great force and violence, the systemic poison is neutralized or destroyed, equilibrium is restored, and all is quiet and serene once more."

Alcoholics constitute a second class, in whom a convulsion or two may have a salutary effect in causing them to quit drinking.

Treatment During an Attack.—During an attack little treatment is necessary. Constricting clothing about the neck should be loosened to lessen the danger of hemorrhagic extravasation into the skin of the face, neck, and conjunctivæ.

When vomiting threatens just after the attack, the patient should be placed on the side to lessen the danger of vomited substances falling back into the larynx. This is rarely a cause of death—one that I have never seen. Gowers mentions a single case of the kind.

If the attacks occur at night, there is danger to life from suffocation. In observations I made in 100 nocturnal epileptics, it was found that 13 per cent. of

them were turned on their faces by the force of the convulsion. Many would have suffocated had they not been relieved from the position. The friends of the patient should always be warned of this danger.

The sleep that so frequently follows an attack should not be prevented. If it is, the patient suffers more from headache and is longer in recovering from the effects of the attack. The prostration that so often follows rarely calls for treatment. It will pass away naturally in time.

For obvious reasons the patient should be laid down if he can be reached in time, not because posture influences the attack one way or another, so far as we know, but to prevent possible injury from falling. It is possible in some cases to prevent the tongue being bitten by placing a piece of cork or India rubber between the teeth.

DRUGS.

Drugs are administered in epilepsy for the purpose of influencing the attacks, either by arresting their occurrence or, failing in this, by rendering them less frequent and less severe.

As yet we are ignorant of any means of suddenly curing the disease, and from what we now know of its etiology and pathology, it seems unlikely that any means of rapid or sudden cure will be forthcoming in the immediate future. This pertains to true epilepsy; not to less fundamental convulsions, some of which are capable of early eradication.

Unfortunately the influence of drugs is transient, making necessary their repeated renewal. The only way in which permanent benefits from them can be obtained is to persist in their administration for years, while some epileptics must take them continuously. *The necessity for prolonged treatment should be made clear to the patient at the outset.* If he is capable of

understanding why this is so, it should be explained to him in order that his co-operation may be enlisted.

The Bromin Preparations.—Since their introduction by Laycock half a century ago, the salts of bromin have enjoyed steady favor as anti-epileptic remedies, notwithstanding the fact that they fall far short of meeting the important indications. Within recent years a marked improvement has been made in the methods of their administration, simultaneously lessening the evils they produce and enhancing their power for good.

Much of the discredit heaped upon the drug in the past has been due to the unguarded manner of its use. Many failures to cure have largely been due to the same cause.

Indefinite suppression of epileptic phenomena must not be mistaken for cure. All drug treatment must be combined with the very essential general treatment I have elsewhere described. We must not feel that any one remedy can suffice; and above all, that we have in the bromids the sum and essence of all drug treatment required.

The Physiologic Action of the Bromids.—The action on the human economy of all the bromid salts is essentially the same. The minor differences of the various preparations worthy of note in the treatment of epilepsy will be mentioned later.

On the nervous system the bromid of potassium— the remedy universal—acts as a distinct motor depressant, coincidently lessening the activity of the intellectual centers. When pushed to an extreme degree it produces apathy, listlessness, lack of interest in surroundings, a dull apathetic expression, and, if carried still further, produces a mental condition not unlike that of primary dementia or stuporous melancholia—a condition from which the patient usually makes a good recovery when the drug is withdrawn.

If pushed too far, death may supervene from acute bromid poisoning. This happened in the case of a boy of twelve years whom I knew, whose parents gave him too frequent doses of a patent nostrum, the essential ingredient of which (as with the bulk of patent epileptic cures) was the bromid of potassium.

Bromid also exerts a sedative action on the spinal cord, lessening reflex excitability in this way.

On the circulation it causes at first a fall in arterial pressure. If given in toxic doses, it arrests the heart in diastole, while its long-continued use results in a weakened and irritable condition of that organ and a general impairment of the circulation, causing cold hands and feet with general blueness of the body.

On the respiration, in ordinary doses, it produces but little effect; in larger doses it acts as a depressant.

On the alimentary canal it often acts disastrously. It irritates the mucous membrane and interferes with the reflex activity of the stomach in a way to check the normal secretion of gastric juice, which impairs digestion.

It causes constipation, heavy coating of the tongue, and foul breath; produces a bad taste and a pasty feeling in the mouth, causes loss of appetite, and not infrequently nausea and diarrhea.

On nutrition it also acts unfavorably in some cases by lessening metabolic changes through depressing the nervous system. It produces unsteady gait; myasthenia, particularly noticeable in the legs; depression and often loss of sexual vigor; forgetfulness and slight degrees of aphasia, usually shown in the misuse of words and the wrong formation of sentences.

On the temperature, in ordinary doses, it has no effect; in larger doses it lowers the heat of the body by depressing the heart's action and possibly by contracting the arteries, a fact apparently established through the experiments of Brown-Séquard, Mairet, and Amory.*

* Ringer's "Therapeutics," 1886, p. 147.

Plate 20.

BROMIC ACNE ON THE BACK.

Bromic acne induced in seven days by 30 grains of bromid 3 times a day. Individual susceptibility to bromid, combined with different states of sensitiveness of the skin, regulate, to a large degree, the amount of acne induced. It is rarely necessary, under modern methods of administering bromid, to create acne to the extent shown in this illustration.

Its effects on the skin in causing acne—widespread, unsightly eruptions called bromism—are universally known. The pustules may turn into circumscribed ulcerations and become phagedenic. Bromid is eliminated very slowly and escapes unchanged. It is found in the secretions, including the sweat, urine, semen, milk, and feces (Wood, Hare, Foster, Brunton, Ringer).

According to Hare,* "Tissue waste is decreased when the economy is under the influence of the bromids." This statement the distinguished writer evidently did not mean to apply to all cases. Some patients may take the bromid in from thirty- to forty-grain doses three times a day for months without impairing nutrition; but it has been my experience that unless care be exercised to combat its toxic effects when long continued, the patient loses weight.

I am not of the opinion that the bromids tend to the conservation of tissue; but feel that the opposite, under ordinary conditions and when the drug is used alone, is more often the case. This opinion is based on a daily observation of its use in several hundred cases of all types and under all conditions during many years and not on any particular scientific investigation made to determine the point. I have noted that most epileptics gain in weight when given a less quantity of bromid or when it is entirely withdrawn.

"The bromids should be reserved as a last resort or as an adjuvant. Cures by the bromid treatment are not to be expected. As a practical fact, if the fits are not controlled by sixty grains of bromid a day, the question is sure to arise as to whether the epilepsy or the bromid is the greater evil. It is a frequent experience to see patients, brutalized by the bromids, go months without fits, but with a loss of mental and physical activity" (Church and Peterson).

* "Practical Therapeutics," 1900, p. 107.

Weir Mitchell noticed among the ill effects of the bromids "irritability, increased irritability or melancholia at menstruation, bad temper, suicidal or homicidal tendencies, and temporary delusions" ("American Journal of the Medical Sciences," 1896).

On the whole, I know of no drugs save those which produce habits such as opium and cocain, that are so universally abused as the bromids. The effects of these drugs on the heart, respiration, gastrointestinal canal, skin, and nutrition, are five points against their use in epilepsy compared to one in their favor, namely, their power of lessening overexcitability of nervous tissue. The question naturally arises, Does the good they accomplish in this way more than counterbalance their injurious effects in other ways? In the majority of cases the answer is, No; in some, Yes.

The secret of obtaining successful action with the bromids lies in the judicious method of their administration, and in carefully selecting each case for their use.

As an emergency remedy in suppressing repeated convulsions that threaten life, like serial attacks or status epilepticus, and used in conjunction with other drugs, the bromids are always valuable. A favorite prescription for this purpose is the following:

R. Potassium bromid .... grs. lx.
   Chloral hydrate .... grs. xx.
   Morphia sulphate .... gr. ⅛.
   Tinct. opii deod. .... ♏iv.
   Aqua ad. q. s. fiat .... ℥j.
SIG.—Give at one dose.

This dose may be repeated in two hours if necessary. The treatment in such cases must be heroic; to temporize may be fatal.

In psychic epilepsy or in the psychic epileptic equivalent it is of little use. In the latter the psychomotor violence is too great to respond to the bromids

Plate 21.

BROMIC ACNE OF THE FACE.

The prolonged unguarded use of bromid may cause not only much disfigurement but phagedenic ulcerations that cause great discomfort and that are often very sluggish in healing. It is seldom necessary to push bromid to the extent required to cause this condition. Rightly administered and watched in its effects, it need not cause these results.

alone. In the various forms of epileptic mania they are practically without virtue. The same is true of their use in most of the epilepsies due to recent trauma. Here they may be especially harmful before the operation by suppressing important phenomena indicative of the seat of the lesion. After operation, and in long-standing traumatic conditions, they are more admissible.

The idiopathic *grand mal* epileptic, of vigorous constitution and good digestion, does best under the continuous use of the bromids. Under the precautions I am about to outline, such patients may take them continuously for years without harm. The size of the initial dose must be regulated by the character of the fit and by the manner in which the drug is given; beyond the third or fourth year, age has but little to do with it.

In most cases it should be given to the point of toleration or to the point required to control the attacks. This can be determined only after trial. Unless the attacks are threatening in frequency and severity, it is best to begin with from five to ten grains three times a day. Prescribed in a watery solution of a definite strength is the best form for giving it alone. In this way a drachm may be made to represent any amount the patient is to have.

The hour of administration is important. A systematic record of all seizures, day and night, should be kept. After a little while, the hour at which attacks are most likely to occur can be determined. If they are more prone to occur at night just after the patient falls asleep, the medicine should be given early in the evening; if they come after midnight, it should be taken on retiring, or about 9 A.M.; if they have a matutinal tendency, as they so often do—occurring just after the patient gets up in the morning—the largest dose should be given before the patient gets out of bed.

Nocturnal epileptics should get larger doses at night than during the day, while the converse is true with those of diurnal habits. The former, if taking from ten to fifteen grains during the day, should have double the quantity on the approach of the seizure period at night, and the same with the latter during the day.

The bromid salts should not be administered in close conjunction with meal hours—an hour before or after is best. They act more promptly on an empty stomach. If habitually taken before meals, somewhat smaller doses may suffice. They should be given in plenty of water, plain or carbonated, such as Vichy or Apollinaris. Their effects are much enhanced when given in this way. The larger the quantity of water taken, the better.

Their administration once begun should not be interrupted unless the patient's condition imperatively demands it. Mothers who are taking the bromids should not nurse their babies. Any severe illness, such as typhoid fever, that saps the patient's strength, should cause their gradual withdrawal.

If the attacks can be held in check by the bromids for two years or more, they may be reduced in quantity at the end of that time. *Sudden withdrawal is never advisable*—to do this is to invite grave disaster in the way of status epilepticus.

The prevention of bromid intoxication is always desirable. Besides the discomfort and disfigurement it entails, it is a telltale of some disease. Except in the rarest instances, it is not necessary to push the drug to the toxic limit.

By beginning with small doses which the patient can assimilate, and being watchful for the first symptoms of its effects on the skin, by keeping the digestion in good condition, the intestinal canal free from the products of putrefactive changes and so lessening the

danger of auto-intoxication, and by habitually guard-
ing against constipation, and easing the disquiet so
often attendant upon the menstrual flow, we can keep
down any marked accessions to the frequency and
severity of epileptic attacks, and so obviate the neces-
sity for the temporary heavy drugging that always
ends in pronounced intoxication.

The chief indication lies in directing treatment to the
numerous conditions that incite epileptic phenomena
to greater activity, and we should not rely on the
bromids to suppress them irrespective of the cause of
their origin. No surgeon would think of applying a
poultice over a splinter to deaden the pain; he would
first remove the cause.

The same principle applies in many cases of epilepsy.
The eliminative system, the skin, kidneys, and bowels,
demand especial attention. The price of failure may
often be found in the neglect of these. A congenitally
weak and hypersensitive nervous system is delicately
enough adjusted to lose its balance (in a fit) under
the excitation of poisons due to imperfect elimination.
Free elimination also greatly lessens the danger of
bromism.

Bromic acne may be further prevented by the use
of *Liquor arsenicalis* given with the bromid, as recom-
mended by Echeverria in 1870; and by daily cold or
tepid baths followed by brisk rubbing, and outdoor
life. Turkish baths in persons of strong vitality are
useful. In other cases, facial massage and steaming
lessen the rash very materially. These are especially
valuable on sensitive skins.

The hypodermic use of the bromid salts is rarely
called for in epilepsy. Occasionally it is employed in
status epilepticus, and usually causes abscess and a
scar. The hypodermic should be given in the but-
tocks or thighs.

The coefficient forms of the bromid treatment include

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the use of other remedies along with the bromids, and the latter under various modifications. It is a common practice to give several of the bromid salts in combination, usually ·potassium, sodium, and ammonium; the two former in ten-grain doses, the latter in five-grain doses. With them may be given small doses of bicarbonate of soda and *Liquor potassii arsenitis*.

When the attacks are ·mostly at night, .belladonna in some form is useful. A good formula includes in each dose two minims of the tincture of belladonna, from ten to fifteen grains of the bromid of potassium, and five grains of chloral hydrate. In the epilepsies attendant upon the menstrual epoch, I have found the bromid of sodium in liberal doses most valuable.

The bromid of strontium has been used with varying success, succeeding in some cases after the other salts had lost their virtue, or had failed in the first instance to give relief. It should be given in from fifteen- to twenty-grain doses in conjunction with smaller amounts of the ammonium and sodium salts. Some patients will bear it in drachm doses without harm. Roche commends it highly; others fail to find any specific virtue in it.

A change from one bromid preparation to another in many cases from time to time proves beneficial.

The hypochlorization method devised by Toulouse and Richet consists in salting the patient's food with sodium bromid instead of ordinary sodium chlorid, the former tasting so like the latter as to be a very acceptable substitute for it. Upon reaching the stomach, the bromid salt is broken up in the same way that sodium chlorid enters the metabolic formation of hydrochloric acid under normal conditions. Entering the stomach at a time of great digestive activity, the sodium bromid performs the function of the table salt, and is so readily absorbed as to promote rapid sedation in practically half the dosage required when administered in the ordinary way.

The disadvantage of this method of treatment lies in specially preparing the patient's food to keep it free from chlorid of sodium. It is easier to do this in an institution than in a private home. Patients of sufficient intelligence may carry a small measure holding the amount of the sodium bromid they should take, and a supply of the latter, salting their food at each meal. Under this method ten grains of the bromid of sodium is as effective as twenty grains given in the usual way, making the plan one of economy.

There is occasion (Richet) in hypochlorization to distinguish the therapeutic from the physiologic effect. According to Féré,* the disadvantage of the latter may outweigh the advantages of the former. While this method has some value, its efficacy is yet on trial. Its adoption is difficult in private homes, while its employment in any event is open to the objection of the bromids generally. No matter how they are administered, constant auxiliary treatment of the gastrointestinal tract and the cutaneous system is required to guard against the evil effects of intoxication.

Special Bromin Preparations.—During the past few years, very excellent results have been obtained from a preparation of bromin in chemical combination with the fatty acids of sesame oil, in which the irritant qualities of the element are entirely absent—bromipin. Its greatest value has been found in feeble or asthenic cases. Given in the form of an emulsion in such cases it is a good reconstructive, usually causing a gain in weight. A good formula is bromipin four ounces; syr. simplex four ounces; spirits of peppermint four drachms; mucilage of acacia, enough to make sixteen ounces. The dose for an adult is two or three tablespoonfuls three times a day after meals.

* "Extrait des comptes rendus des sciences de la Société de Biologie," Feb. 28, 1903, t. IV, p. 279.

Among its advantages are these: It may be given hypodermically in status epilepticus without the risk of abscess, a desirable form of administration when the patient is in a comatose and critical condition as the result of repeated seizures; its entire freedom from irritating effects upon the gastrointestinal canal; and its failure under maximum dosage to cause bromic acne.

In children it may be employed to great advantage combined with linseed or Irish moss tea, and given in the form of enemata (Rahn). In infants the dose is one gramme for each month in age; in children the dose is from ten to fifteen grammes. Instead of using a 10 per cent. solution for this purpose, one of 33 per cent. may be used when rapid sedation is required.

When given in the form of enemata, the patient should lie on the left side with legs drawn up; a rubber cannula is introduced about twelve centimeters and the fluid slowly injected. It may be used in this way for two or three weeks, night and morning.

The chief obstacle to the use of bromipin at the present time is its cost, which is five times that of the bromid salts.

How the Bromin Preparations Act.—Notwithstanding the length of time that we have known the bromids to lessen the tendency to spontaneous discharges, we have no precise knowledge of the method of their action. That they act in consequence of the presence of the element bromin, there can be no doubt. Bromid is said to cause contraction of the small vessels; but if it has this effect, it is not likely that its influence on epilepsy is thus produced (Gowers). It is evident that it must be carried by the plasma to the nerve structures of the brain. There it seems to restrain the undue tendency of the constituents of the nerve substance to escape to combine with the oxygen of the plasma. If it acts in this way, it is not known just how. It may do so through the mere effect of

its presence, or by yielding its bromin to combine with constituents of the nerve elements. All knowledge along this line is as yet speculative.

Bromin is a strong disinfectant and it may eventually be found that its value in epilepsy is due to some extent to its power of neutralizing morbific agents in the blood. This seems fully as tenable a theory at this time as that which assumes that the presence of bromin in the nerve structures serves to reduce their morbid affinity for oxygen.

Whatever its mode of action, it is often feeble and uncertain and always transient, while its elimination from the system is not accomplished under two to three weeks, so that while ·its beneficial effects are brief, its prejudicial effects are often indefinitely sustained.

If epilepsy (as it is now so generally believed) is due to "an abnormal readiness of the cells of the cortex for action," and if the bromids lessen their tendency to discharge—no matter how—we can see that the reason they so often fail to effect a cure is that they do not remedy an elemental cytologic defect, but merely lessen or suppress nervous hyper-excitability. But even this is valuable, for, during suppression, time is given for more tedious but more lasting forms of treatment to become of permanent service.

The Opium-bromid Treatment.—Opium was recommended in epilepsy by Paracelsus in the fifteenth century, and by Crato and Quercetanus in the sixteenth century, while De Haen reported a patient cured by a large dose of laudanum, a sequence of doubtful credibility (Gowers). Opium alone is seldom used in epilepsy. While its power to suppress the attacks temporarily may not be questioned, its danger in other respects is great.

In 1893 Flechsig originated a method of employing

opium and bromid in conjunction in epilepsy. The treatment consisted in keeping the patient in a state of mild opium narcosis for six weeks, beginning with a small amount daily and increasing it to fifteen grains at the end of that time. The opium is then suddenly withdrawn and bromid substituted in from 120- to 130-grain doses daily for two months, after which the amount is diminished gradually to thirty grains a day.

Flechsig recommended this treatment only in chronic cases in which the bromids alone had failed, and cautions that the patient be carefully watched during the whole course of treatment.

In my experience the plan was of little use. Many patients of the type in which it is safe to use it could not endure such heroic medication. Out of twelve submitted to it, two had to be withdrawn before the end of the second week on account of the extreme stupefaction. In the remaining ten, six showed no improvement at the end of the bromid course, two had their attacks temporarily suppressed, while in the remaining two they were lessened in frequency and severity. All of them were practically incapacitated during the treatment through mental stupor and unfitness for physical exertion.

The verdict of those who have tried the method is almost uniformly unfavorable, including Donath, Bohme, Landois, Luske, Kathe, Homen, Liehen, Warda, and Bratz. The latter in the treatment of forty-six cases met with three cases of status epilepticus and several cases of severe mental depression (Gowers). Some writers report more favorably.

In my experience the attacks of epilepsy can be materially reduced in this way in almost every case that has shown itself obstinate to the ordinary bromid plan (Collins).

The treatment of Flechsig is of value for many

patients, especially in old and obstinate cases when all other agents have proved ineffectual (Church and Peterson).

In this connection the occasional effects of morphia in epilepsy should be mentioned. Bad effects are apt to follow when the period of epileptic coma happens to coincide with the sleep induced by the morphin. Gowers mentions a case in which an epileptic about to undergo a surgical operation was given a quarter of a grain of morphin to relieve great pain. As the drug was beginning to take effect, the patient had a seizure and immediately passed into profound coma that rendered artificial respiration necessary for an hour.

Codein in conjunction with the bromids has been used quite successfully in lessening the frequency and severity of attacks in chronic cases. Its failure to arrest secretion in the respiratory and intestinal tract is a point in its favor over opium.

Many epileptologists deny the value of codein in epilepsy. It cannot be claimed to have any curative properties. In a feebler way than opium it tends only to suppress epileptic phenomena and may be tried after other remedies have failed.

It is never safe to employ opium in any form in epilepsy unless the patient is under a physician's constant care. Disastrous consequences may develop in any case.

Borax.—Next to the bromids many believe that borax (sodium borate) enjoys favorable distinction as an anti-epileptic remedy. It was first suggested and used by Gowers in 1879,* who has since remained a believer in its efficacy. It may be given in combination with bromid and deserves a trial in obstinate cases. The dose is from fifteen to thirty grains three times a day when given alone. If given in combination with bromid, the dose may be larger.

* "Goulstonian Lectures," "Lancet," 1879, Vol. I.
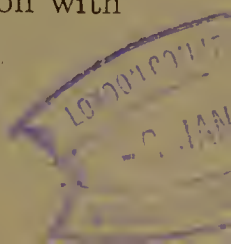
In some patients it acts unfavorably by causing diarrhea with dysenteric evacuations if the dose is too large at the beginning. A reduction in the quantity causes untoward symptoms to disappear. It has been given as high as 120 grains a day, though not for any length of time.

Another complication likely to appear is psoriasis, though not under several months' administration. The addition of arsenic to the borax will cause the psoriasis to disappear. In this class of cases it is only after the bromid has failed that we need employ borax. Gowers mentions an obstinate case that yielded to borax and tincture of gelsemium in combination after numerous other remedies had failed, the former being given in twelve-grain and the latter in five-minim doses.

Mairet* sums up his conclusions concerning the use of borax in epilepsy as follows:

1. Borate of soda may have a real utility in the treatment of epileptic attacks, which it may diminish or even entirely suppress for many months.

2. Borate of soda succeeds better in symptomatic epilepsies than does the bromid of potassium.

3. Bromid of potassium acts better on the epilepsy neurosis.

Borate of soda was tried at the Colony in ten carefully selected cases, all of an obstinate character. The initial dose was ten grains three times a day, increased later to fifteen grains. In three cases only was there a diminution in the number of seizures. Upon the gastrointestinal tract its evil effects were noted in nearly all cases at one time or another. Its cumulative tendency gave use to persistent attacks of furunculosis, which led to its abandonment.

Chloral hydrate is of little use in the treatment of epilepsy ordinarily. I have found it of greatest value

* "Le Progrès médical," No. 41, Oct. 10, 1901.

in cases of mental disturbance (epileptic mania), and in obstinate insomnia, which is not often encountered in epilepsy independent of disturbed mental states. Some patients who show a psychic aura several days prior to the attack, who are mildly excited, loquacious, eyes unusually bright, pulse quickened, and face flushed, are good subjects for its use. It is best given with bromid acacia, simple syrup, or *syrupus pruni virginianæ* as adjuvants. Its value in nervous insomnia may be enhanced by the addition of one-eighth or one-sixth of a grain of morphin to the dose.

Rarely we may find an epileptic exhibiting for weeks at a time the pre-convulsive symptoms enumerated above. Three cases of the kind have come under my care. Chloral hydrate in eight- or ten-grain doses two to three times a day was more effective in them than anything else.

In serial attacks and in status epilepticus cases that resist all other measures, chloral hydrate is often of great service. The contingency being great, the remedy at times must be used heroically. In the first stages of status epilepticus it may be administered by the mouth. When coma supervenes with the attacks continuing, it should be given by the rectum in conjunction with bromid. The moment it is noted that the sedation is controlling the attacks, its reduction should be begun. It is always dangerous to give chloral when cardiac failure is imminent. If a first dose of thirty grains is not effective in serial attacks or status epilepticus, a second may be given in three hours, the heart permitting.*

Amylene hydrate was tried by Weber in seventy cases of long standing and equally divided as to sex. Most of them received the drug in from half to two drachm doses daily. Eighty per cent. showed no marked variation in their convulsions; fifteen per

* See chapter on Status Epilepticus.

cent. showed a decided increase; while five per cent. only showed a decrease.   Except in the last five cases, all showed disturbance in general health, drowsiness, mental sluggishness, and digestive disorders, which led to the drug being discontinued.   A diminution in attacks occurred in ten per cent. of the same cases under resumption of bromid.

Wildermuth speaks favorably of amylene hydrate in cases in which bromid has failed, provided the course of treatment is short.   In from four to six weeks the evil effects noted by Weber begin to appear.   It occasionally acts better in status epilepticus than chloral, and according to Wildermuth is often effective in the eclamptic attacks of children.

Nitroglycerin is serviceable in cases of senile epilepsy with evidences of arteriosclerosis; not so much on account of its effects upon the attacks, as in lessening the extreme discomfort that not infrequently precedes and attends them in such cases.

A man, sixty-five years old, of voracious appetite, plethoric, and with whip-cord arteries, suffered the most distressing pain in the precordial region for hours before an epileptic convulsion.   He was given $\frac{1}{100}$ of a grain of nitroglycerin three times a day, his diet and exercise regulated, and at the end of three months he had experienced a most positive relief—his convulsions (which were less in number) being attended with none of the former painful manifestations.

Nitroglycerin acts rapidly and the effects are of short duration, requiring that it be given every three or four hours.

It may also be employed in conjunction with bromid in the *liquor trinitrini* of the pharmacopeia, a one per cent. solution of nitroglycerin in alcohol.   To insure its stability, it must be rendered acid, which can be done by adding from ten to fifteen minims of dilute hydrobromic acid to each dose.

It is also often useful in the periodic headaches that accompany epilepsy. It is also of great value in combination with bromid in cases that show a feeble circulation, slow pulse, coldness, and pallor of the extremities.

Gowers speaks of its good effects in attacks of momentary unconsciousness in children. It has long been my belief that such attacks were not infrequently associated with pathologic conditions in the circulatory system. I recall three boys between nine and twelve years in whom marked improvement followed the exhibition of heart tonics. All had normally a very slow pulse. In two of them the beat, which I had opportunity to count, fell from 60 to 48 a minute at the beginning of the attack.

Chloretone.—The results of chloretone in epilepsy are not unlike those of Flechsig's treatment. Weeks tried it in six chronic cases, with the result that when pushed to as high dosage as seemed safe, it lessened the frequency and severity of the attacks in most of them. Coincidently it caused marked somnolence, headache, and vertigo. Pallor was noticed in nearly all who took it. Weeks advises that the patient be brought under its influence as rapidly as possible, after which the dose should be gradually reduced. In most cases six grains three times a day is sufficient to prolong the effects obtained.

Zinc is among the older remedies used in epilepsy. It was prescribed more than a century ago. In the pre-bromid days it took first rank as a therapeutic measure and was praised by Herpin, who is reputed to have effected so large a percentage of cures.

Its action is analogous to bromid, though less powerful. It lessens reflex action, influences the functions of the cerebral hemispheres, and generally acts best in the milder cases. Of all its salts, oxid, lactate citrate, sulphate, and bromid, the former is most

generally used. Individual susceptibility to it varies greatly. Some patients can take eight or ten grains with impunity; in others, two or three grains cause nausea. It is best administered in pill form. The oxid salt is doubtless converted into chlorid in the stomach, which is too slowly absorbed to become an irritant.

It was tried at the Colony in ten cases in conjunction with salol. In all of the cases selected there was muscular tremor or marked nervous irritability. After a fair trial, in a small percentage of the cases there were fewer attacks, but the decrease was not maintained on the withdrawal of the drug.

Urethan has been praised highly by Jacobi as a remedy in epilepsy. I selected six cases of the idiopathic disease for its trial; three of the patients were subject to diurnal, three to nocturnal attacks. In the diurnal cases twenty grains were given each patient three times a day; in the night cases twenty grains morning and noon, and forty-five grains at night. The trial extended over thirty days, during which notes on temperature, skin, pulse, respiration, reflexes, circulation, headache, and mental condition as affected by the drug, were kept. In no case did it act harmfully in any respect. One patient had no attacks while taking it, having had seven during the previous month; two had their attacks reduced to two-fifths their former frequency; three remained unchanged. I regard *urethan* as worthy of trial in cases in which the bromids give no benefit.

Solanum carolinense was introduced by Napier of South Carolina a few years ago. It was given to ten men and eight women at the Colony for a period of two months. In six of them there was a considerable reduction in the number of attacks during the period of treatment. Collectively they had 133 attacks during the two months preceding the use of the drug,

and 95 during an equal period while taking it. In one patient the improvement was sufficiently marked to warrant its continuance over a period of eighteen months. During four months prior to treatment he had 18 *grand mal* attacks. The first month under treatment, he had none; the second, five; the third, three; while during the following fifteen months he had but one. The drug was then discontinued, with the result that during the next five months he had seven seizures.

Among the remaining eleven cases one patient died in status epilepticus, while ten experienced no beneficial results.

A second trial of the drug in eight selected cases failed to produce results as good as those above. My experience with *solanum carolinense* leads me to believe that we have in it an effective remedy in selected cases. Those taking it should possess a strong physique, good digestive powers, and show as little mental impairment as possible. Severity of attacks makes no difference. It is given in the form of a fluid extract in doses of half a drachm to a drachm three times a day.

Simulo.—The investigations of Eulenberg ("Therapeutic Gazette," Oct. 15, 1888) directed attention to the use of *simulo* in epilepsy. It is made from a South American plant of the hyssop family and is given in the form of a tincture in two- or three-drachm doses three times a day. It has attained considerable prominence in cases in which bromid was not well borne alone, and is often valuable when prescribed in conjunction with it.

"I would say of *simulo* that it deserves trial in most cases; that it is perfectly harmless, which cannot be said of the bromids, belladonna, and some other drugs; . . . . that in most cases it has no effect at all" (Peterson).

It was tried for one month in ten cases under my care, improvement resulting in two of them. During the month preceding its trial, these two patients had 194 seizures; during treatment, 123; and during the month following, 163. The remaining eight patients had 80 seizures in the month before treatment, 109 during it, and 114 during the month following. These results were obtained under small doses to test its constitutional effects, which were *nil*. On increasing the quantity to two and three drachms three times a day, there was still failure to produce any toxic effects, while a further reduction in the attacks was induced.

On the whole, *simulo* and *solanum carolinense* are well worth trial in cases that are over-susceptible to bromid intoxication, or in which bromid does not lessen or suppress the attacks. They have merit in being comparatively harmless under anything like rational administration.

Trional.—The very general advocacy of this drug in epilepsy a few years ago led me to try it in 7 nocturnal cases, the trial lasting thirty days, the patients being kept thoroughly under its full effects all the while. It was first administered in twenty-grain doses followed by thirty grains of effervescing Vichy salts in a glass of hot water. The Vichy so enhanced its action that ten-grain doses of the trional sufficed to keep its full physiologic effects in view. Beyond some dulling of cerebration, vertigo, and general muscular weakness, no evil effects were noted; nor did it have any appreciable effect on the disease itself.

In Case I there were 114 attacks before treatment, 96 during treatment, and 99 after (each number in thirty days); in Case II, 8 before, 4 during, and 6 after; in Case III, 5 before, 11 during, and 9 after; in Case IV, 4 before, 4 during, and 6 after; in Case V, 51 before, 59 during, and 59 after; in Case VI, 35 before,

18 during, and 17 after; in Case VII, 5 before, 3 during, and 3 after.

I mention these negative results particularly to call attention to the apparent inertness of a remedy that exercises so great an influence on the brain. Notwithstanding its ability to induce profound sleep, it showed no power to check the convulsions.

Iron.—The administration of iron in epilepsy has been opposed by many distinguished authorities, including Hughlings-Jackson and Brown-Séquard, who held that it increased the attacks.

Indiscriminately used, this may be true, but I have found its action beneficial in certain cases. Its use is especially advised in chlorotic or anemic young women who develop epilepsy, or whose epileptic attacks become accentuated at puberty. I have also noted its good effects in cases of pernicious anemia in older persons. If it is *substituted* for bromid, the effect of the cessation of the bromid must not be ascribed to the influence of the iron.

Gowers believes its beneficial influence is not confined to its hematinic effect; that it has a specific action on the nerve centers similar to that caused by other metals. Meyer and Williams found that it had such action by injecting the tartrate of soda and iron into the blood of animals, with the result that it caused paralysis of central origin, the excitability of the muscles and peripheral nerves remaining intact.

In an unmarried epileptic woman of twenty-eight years under my care, who suffered from marked anemia, iron was the only remedy that checked her convulsions.

It may be given in the form of the tincture perchlorid in from ten- to fifteen-minim doses three times daily, or in pill form as follows: Ferric ferrocyanid $\frac{1}{2}$ of a grain, extract of belladonna $\frac{1}{8}$ of a grain, extract of gelsemium $\frac{1}{8}$ of a grain.

I have had uniformly good results from iron pepto-nate and manganese in combination (Pepto-Mangan, Gude's) in cases demanding hemic reconstruction. To add small doses of strychnia nitrate—from $\frac{1}{30}$ to $\frac{1}{60}$ of a grain—to each dose of the Pepto-Mangan often gives still more satisfactory results.

The coal-tar derivatives, including antipyrin, phen-acetin, and acetanilid, especially the latter, for some time enjoyed what I feel to have been an unwarranted popularity in the treatment of epilepsy. I carefully tested the value of each in a large number of cases selected to meet the physiologic actions of the drug. It could not be ascertained that any of them exerted any beneficial influence over the seizures. They were occasionally found useful in allaying post-convulsive headache. In epilepsy as elsewhere their continued use is inadvisable on account of the manner in which they affect the heart.

Chloroform.—"Whatever opinion theoretical writers might have advanced on the use of chloroform in epilepsy, I believe that few practical physicians after having tried it are willing to place reliance on it," wrote Echeverria a third of a century ago.

After an extended experience with the drug in this respect, I am of the same opinion. In a few cases of status epilepticus I have seen it check the convulsions; but I have seen it more frequently fail to do so. In one case in which the patient had 549 seizures in two days, profound chloroform narcosis was kept up nine hours altogether, two hours at one time, two at another, and five the last time. All the attacks began in the right thumb, next involving the right arm, then the right leg and right side of the face. During the chloroform narcosis they did not extend to the oppo-site side, otherwise they did. This was the only favorable effect obtained. Death occurred after the five hundred and forty-ninth seizure.

Moreau has stated that in *vertiginous epilepsy* chloroform may prove fatal (this possibly refers to syncopal attacks due to heart disease). Fox noted, in using it to detect simulation, that it increased the attacks in three cases. Tosquinet* is of the opinion that it induces the paroxysms. Echeverria is unable to recall any epileptic suffering from successive fits who was relieved by inhalations of chloroform or ether.

Six patients presenting gastric aura and showing some debility were selected at the Colony and given chloroform and glycerin internally for several months; the former in ten-minim, the latter in one-drachm doses, with satisfactory results. The attacks were decreased in frequency coincident with improvement in the physical condition. A very small proportion of cases are eligible for such treatment. The essentials for it include a state of general debility, and an aura that implicates the pneumogastric nerve.

Electricity has been advocated in epilepsy (Féré, Althus, Fischer), either in the form of galvanization of the sympathetic or of the head, the current being passed obliquely from the frontal region to a point diametrically opposite at the nucha; or, as Althus recommends, transverse galvanization of the mastoid apophyses. Others advise general faradization.

In the great majority of cases, the philosophy of such treatment is no more apparent than that of treating epilepsy with electric light rays in a confined room, as Hughes advised. Direct applications of the electric current may indirectly benefit epilepsy in a very small number of cases by benefiting the patient's general condition.

Hydrotherapy is beneficial in selected cases. It often does good by relieving the effects of bromid intoxica-

* "De l'inspiration du chloroform comme moyen de constater l'épilepsie," "Archives Belges de Médecine," t. XII.

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tion. It can only benefit the epilepsy through the favorable modification it induces in the patient's general condition. The methodical tests made by Fleury confirmed this fact.

Any form of hydrotherapy that renders more regular and active the peripheral circulation will prove beneficial in epilepsy. For details of such measures the reader is referred to the last edition of Baruch's admirable work on this subject.

The serum treatment of epilepsy was carefully tried by Ceni* under methods elaborated by himself. Ceni's experiments were based on the opinions of Evans, Regis, Chevalier, Lavause, Jacobson, Féré, Voisin, D'Abundo, Herter, and others, to the effect that some toxic irritating cause, probably of a biochemic nature and elaborated by the organism, must be of capital import in the determination of the epileptic fit.

Ceni first tried to ascertain if epileptic blood serum, which was assumed to contain a specific poison, would not in time introduce into the system some property of therapeutic value in earlier and less severe cases. The results were not encouraging. He then tried to render epileptics less resistant to the actions of poisons circulating in them, through progressive doses of the same serum—to establish immunity.

Without going into the details of the results as set forth in Ceni's paper, it may be said that they differed in no essential respect from those obtained under almost any new method that so jealously guards the patient's welfare in general ways while it is under trial.

The influence of concurrent diseases in epilepsy has often been called to attention. It appears that Hippocrates spoke of it first in connection with malaria.

* "Serotherapy in Epilepsy," Craig Colony Prize Essay, "Medical News," March 8 and 15, 1902.

Marandon de Montyel and Maurice Didé * thoroughly investigated the relationship between malarial infection and epilepsy in several cases.   While they do not deny an apparent improvement under such an infection in some cases, they cite fourteen instances in which it was followed by grave results.   In no instance were they able to say that the epilepsy disappeared with the appearance of malarial fever.   In two cases seizures came on coincidently with the rise in temperature; in others, during the intervals between the malarial paroxysms.

These writers agree with Féré that the chances for improvement are too uncertain to warrant the utilization of bacterial products in the practical therapeutics of epilepsy, as has been proposed.

Bourneville † reports an epidemic of typhoid fever among children at the Bicêtre, seven of them being epileptics. "All seizures," he states, "were suspended during the attack of fever. This inhibiting effect persisted after recovery, only one or two attacks having been since noticed among them all."   The length of time of freedom from the attacks is not given.   Sometimes typhoid fever causes status.

Clark and Sharp ‡ report five cases of measles and seven of erysipelas occurring in epileptics at the Craig Colony, summarizing their conclusions as to the effects of the two diseases on epilepsy as follows: "Our deductions in these cases are that *in not one instance was the* pre-existing epilepsy favorably modified for any great length of time."   (Italics theirs.)

In cases that underwent favorable modification it was slight and of short duration.   It seemed clear that the status epilepticus in one case was due to the effects of the measles.

* "Revue de médecine," Dec. 10, 1899.
† "Progrès. médical," Paris, Sept. 2, 1899.
‡ "Medical News," Dec. 1, 1900.

In six patients under my care who contracted diph-
theria, the attacks for three months before such illness,
and for a corresponding period after, were as follows:

Case   I, 19 before, 45 after.
Case   II, 20   "   19   "
Case III, 19   "   4   "
Case IV, 7   "   15   "
Case   V, 43   "   50   "
Case VI, 25   "   15   "

Ebstein has called attention to the associated
occurrence of diabetes and epilepsy, but does not
attempt to explain any relationship there may be
between them.

The Relief of Eye-strain in Epilepsy.—A few ophthal-
mologists (Gould, Stevens, Ranney) claim that the
relief of eye-strain in epilepsy not infrequently cures
the disease.   Some, like Stevens and Ranney, believe
in operation for the correction of imbalance of the
ocular muscles; others, like Gould, think operation is
inadvisable, and seek to correct the trouble with
glasses only.

I am unable to recall a case of epilepsy in all my
experience in which I felt that defective ocular condi-
tions alone caused the disease.   Several patients have
entered the Colony whose eye muscles had previously
been cut without any improvement in their epilepsy.

Gould and Bennett made a most careful scientific
test of the possible influence of eye-strain upon the
etiology and cure of epilepsy at the Colony in 1902.
I quote in part from their preliminary report.*

"The examinations of the eyes were begun on
August 18, 1902, and continued for five days.

"We examined in all seventy-eight patients, the
youngest ten, the oldest fifty-nine years of age, the
majority being young or middle-aged adults.   Of

* "Eye-strain and Epilepsy: A Preliminary Report," "American Medi-
cine," Vol. IV, No. 11, pages 416, 417, Sept. 13, 1902.

these seventy-eight, two were excluded because of organic diseases of the eyes which rendered them useless for the purposes of the tests in view. Five more were excluded because of the impossibility, due to psychic or ocular amblyopia, of diagnosticating the ametropia. This left seventy-one cases. Of these, three were excluded because the ametropia was of so low a degree that it was thought negligible. These patients needed no glasses, either for the relief of ocular conditions or of reflex results. Only about 4 per cent., therefore,—three out of seventy-one cases,— seemed to us to have eyes so near normality of optic conditions that they required no further attention.

"Our tests, therefore, concern sixty-eight cases,— thirty-five men and thirty-three women. These were chosen for us by the superintendent regardless of all conditions of epilepsy, age, etc., except that we requested that only patients be given us who were sane and who could read.

"The errors of refraction were estimated only after thorough paralysis of the accommodation by means of homatropin and cocain. Dr. Bennett diagnosticated the muscle-imbalance, made the ophthalmoscopic examinations, and estimated the refractive errors objectively by means of the retinoscopic method. Dr. Gould made the subjective refraction and accommodation tests, and dictated the prescriptions. The subjective tests were in all cases those finally relied upon when the patients' answers could be trusted, and the results seemed the more accurate.

"The following table of the sixty-eight cases shows:

Number of Cases.	Approximate Per Cent.	
13	20	Myopic or compound myopic astigmatism.
54	80	Hyperopic or compound hyperopic astigmatism.
33	50	Unsymmetric astigmatism.
15	22	Normal acuteness of vision (with correction).
23	34	Moderately subnormal acuteness of vision (with correction).

Number of Cases.	Approximate Per Cent.	
30	44	$\frac{20}{40}$ vision or less (with correction).
3		Regular isometric, compound astigmatism.
1		Simple regular astigmatism.
1		Simple hyperopia.
0		Simple myopia.
9		Absolutely isometric, *i. e.*, about 77 per cent. had anisometropia.

"The muscle-imbalances for any high or compli-
cating significance were unexpectedly absent. In-
deed, in but one case did we think them worth con-
sideration, so far as final correction was concerned.

"The astonishing fact, and one that we think de-
serves most serious attention, is the enormous pro-
portion among these patients of cases of injurious
astigmatic and anisometropic defects; in sixty-seven
out of sixty-eight cases there was astigmatism; and it
is most noteworthy that about half of the entire
number of patients had unsymmetric astigmatism, a
defect which almost inevitably produces the most
injurious results upon cerebral and assimilative func-
tion. This terrible incidence of unsymmetric astig-
matism in epileptics is, we judge, twenty or more
times as great as in ordinary patients. We do not say
that these high and most injurious ametropic defects
caused the epilepsies of these patients. That can only
be determined in the future by the careful records of
seizures to be kept and compared with those of the
past. If none of the patients is cured by the relief
of eye-strain it would still not disprove the theory
that in a certain number the eye-strain might have
been the initial cause."

The sixty-eight patients were carefully fitted with
glasses by an expert optician, were placed under the
care of a physician who saw them daily to insure
proper wearing of their glasses, and a record was
kept of all attacks, day and night, for a year after.
The following table gives the results in detail in all
presenting an unbroken record in the period of treat-

ment for three months before and three and six months after wearing glasses.

The results at the end of the year were not far different from those at the end of six months.

TABLE SHOWING RESULTS OF RELIEF OF EYE-STRAIN IN EPILEPSY.

	MALES.				FEMALES.		
Case No.	Number of Attacks during Three Months Prior to Wearing Glasses.	Number of Attacks during Three Months Following the Wearing of Glasses.	Number of Attacks during Six Months Following the Wearing of Glasses.	Case No.	Number of Attacks during Three Months Prior to Wearing Glasses.	Number of Attacks during Three Months Following the Wearing of Glasses.	Number of Attacks during Six Months Following the Wearing of Glasses.
1	69	29	29	1	57	59	119
2	2	4	22	2	9	14	25
3	12	8	8	3	1	0	0
4	7	13	28	4	5	10	19
5	6	2	6	5	7	7	24
6	14	21	36	6	79	16	204
7	11	112	138	7	22	4	41
8	23	11	31	8	1	1	3
9	23	8	19	9	0	0	0
10	0	9	9	10	0	0	0
11	0	1	3	11	0	3	9
12	3	3	7	12	202	105	344
13	9	16	20	13	14	14	17
14	6	4	11	14	0	0	0
15	0	0	0	15	4	10	14
16	4	1	3	16	36	42	73
17	3	4	8	17	6	0	7
18	306	222	400	18	0	0	0
19	0	0	0	19	17	23	36
20	24	24	43	20	7	4	8
21	3	3*	3	21	1	3*	0
22	33	33	50	22	26	32	64
23	11	3	23	23	48	59	89
24	24	14	29	24	50	59	83
25	35	32	35	25	22	14	24
26	74	85	185	26	17	50	99
27	2	6*	6	27	1	6	12
28	4	10	17	28	12	21	40
29	33	56	107	29	3	2	6
30	0	0	6	30	2	8	17
31	25	31	50	31	7	11	17
				32	2	2	3
				33	12	13	29
Total	766	765	1332	Total	670	592	1426

*These cases passed from under observation at the end of the first three months' period.

The results of this experiment so carefully made were not encouraging. The nearest approach to a cure was in case No. III (male). This patient had no attacks in June, eight in July, and four in August, the three months preceding the use of glasses. He began to wear them September 1, 1902, and had eight attacks in the next twelve months, four in September and four in October, 1902. His attacks were *grand mal*, always severe, and universally began with a bilateral arm aura, both hands finally being jerked above the head some time before the fit.*

Some patients (four or five) declared that the use of glasses had benefited them in some way, mostly in the relief of headache.

The totals show that the thirty-one males had 766 seizures during the three months prior to the wearing of glasses, 765 during the first three months after, and 1332 during the first six months after; that the thirty-three females had during three similar periods, 670, 592, and 1426 seizures.

* After going 14 months without an attack, they recurred in January, 1904, as severe as ever.

CHAPTER XV.

THE SURGICAL TREATMENT OF EPILEPSY.

Types of Epilepsy Suitable for Surgical Intervention. Jacksonian and Grand Mal, Idiopathic, Organic, Traumatic. Clinico-pathologic Guides to Operation. Technique of Trephining. Technique of Abdominal Section with Indications for the Performance of Each. Results.

THE presence of epilepsy being established, there are two essential preliminaries to be observed before undertaking any operation upon the brain for its possible relief:

1. The determination of its type and cause.

2. The determination of the point on the cranium at which the operation is to be performed.

Both embrace questions of the utmost importance, the determination of which rests with the physician before the surgeon can be called upon to operate.

In some forms of epilepsy surgical intervention is permissible, often valuable, less often curative; but in others it is wholly unjustifiable.

At the outset we exclude from surgical consideration, so far as operations on the brain are concerned, *petit mal*, *psychic*, *hystero-epilepsy*, *serial attacks*, and *status*. Rarely we may find accidental and isolated occasions for operating on some of these, but even so, the principles and methods applicable in other types apply fully as well as in these.

The surgical treatment of pseudo-epileptic convulsions that follow peripheral irritations will be mentioned later.

Excluding the inoperable ones enumerated above, we have but two varieties left for possible surgical treatment, and these are *grand mal* and *Jacksonian*

epilepsy. The latter, as we have already seen, appears as a distinct affection under one form, impossible ordinarily of being mistaken for any other. The former is encountered from an etiologic standpoint under three types,—idiopathic, organic, and traumatic.

Traumatic grand mal epilepsy comes more often to the operating table than any other type of the disease. We have previously noted that in 814 male and 509 female epileptics carefully studied, trauma was the ascribed cause in $8\frac{1}{2}$ per cent. of the former and $3\frac{1}{2}$ per cent. of the latter. Male epileptics far more often than female epileptics are therefore candidates for surgical intervention. It was also observed that in seventy-three out of eighty-eight cases in both sexes the trauma was received before the twentieth year; while reference was likewise made to the manner in which such injuries were most frequently received.

It cannot be said that the trauma was the sole cause of the epilepsy in all cases, but it was unquestionably a contributing factor when it did not act alone.

The type of cases in which surgery most often fails to give satisfactory results, even when it is done at the proper time, *i. e.*, immediately after the receipt of the injury, are those in which the injury was a part of the cause only, often the minor part, the chief cause lying in the patient's strong epileptic predisposition. In many such cases the epileptic state is divided from the non-epileptic state by a thin partition which the injury destroys and which no operation can restore. It is essential that we understand this fact before operation is undertaken. If it is optional with us to operate or not in cases of unfavorable heredity, it is generally better to err on the side of refusal than to proceed in the face of such an obstacle and expect good results to follow. This is true as a

general condition; in exceptionally rare instances oper-
ation may be justifiable, no matter how unfavorable
the patient's family history may be.

We have elsewhere noted (Prognosis) the good
results that often follow medical treatment in the
worst hereditary cases. The same principles are
applicable here, namely, the disease being so largely
due to heredity and so likely to follow minor indis-
cretions, the patient is doubly careful to guard against
the infinity of irregularities that often bring the
paroxysms to light. On the other hand, one must not
forget that the after-treatment of surgical cases has
been growing in importance of late years, and that
an operation often serves to enhance the opportunity
for the more effective usage of the former.

Roswell Park very aptly set forth this point when
he said: "Operation, when indicated and undertaken,
should be regarded as a first measure to be followed
and often preceded by others looking to a correction
of all faults of diet, elimination, etc. Long-continued
attention to these matters is the price of suc-
cess."

CLINICO-PATHOLOGIC GUIDES TO OPERATION.

If the epilepsy is supposedly due to trauma we first
look for scars on the cranium. These may be of the
scalp only and show no evidence, such as depression,
tenderness on pressure, etc., of fracture of the skull.
They are most often found over the motor areas. "As
convulsions play so minor a rôle in the acute symptoms
of fractures at the base, we would hardly expect epi-
lepsy to be a consequence of that injury, and it does
not seem to be one. In 826 cases at the Craig Colony
for Epileptics there was only one in which there was
any suggestion of a fracture at the base."*

* Pearce Bailey, "Fracture at the Base of the Skull, Neurological and
Medicolegal Considerations," "New York Medical Record," May 16, 1903.

As a rule, the epilepsies that follow injury to the frontal lobes demand early treatment for good results. When neglected, they produce early mental death. The absence of depression at the site of the scar does not preclude the possibility of fracture. There may also be subdural or epidural hemorrhage without fracture, either causing epileptiform convulsions that in time may pass, so far as results are concerned at least, into the true disease. Organic epilepsies of this kind either come on just after the injury or they may be delayed some time, the patient passing in the meanwhile through a serious illness, having "brain fever," some degree of paralysis which corresponds with the site of the clot or injury, a high temperature, great prostration, delirium, and often a series of acute convulsions that may be so severe and so frequently repeated as to run into status.

The sooner the operation is performed in these cases, the more hopeful the prognosis of recovery. As a rule, good recoveries are made from the operation when the patient's stamina is good and when he does not represent an epileptic ancestry.*

"A student," reports Keen,† "was injured in a football game. When I first saw him, three days after the accident, I found that he had been unconscious for a half hour after the accident, and ever since then had complained bitterly of headache which he located always in the forehead. Mentally he was very dull, though not comatose. His pulse was slowed down to 52 instead of 72, the normal. Soon after the accident he began to develop convulsions, first in the right leg. afterward in the right arm also, the right arm being

* In speaking of such cases, we have especially in mind the great importance of early treatment to prevent the patients from becoming true epileptics. Recent convulsions due to trauma do not belong to the class of essential epileptics.

† "Philadelphia Medical Record," Dec. 13, 1902.

finally the chief seat of the convulsions.   When they were severe, they involved the left side also.   The face was never involved.

"In six hours and a half after I first saw him he had twenty-four of these attacks, all limited to the right arm.   (This is a clear illustration of Jacksonian epilepsy, needing only the more permanent establishment of the localized convulsive attacks.   The author.) They were not attended with any loss of consciousness. They exhausted him very greatly, especially when they were excessively severe.   Several times it was necessary to give him chloroform.

"There was no fracture of the skull.   The only physical evidence of any injury was a very slight bruise at the outer end of the left eyebrow.   Had I seen this patient before 1885, I should have been unable to explain why the spasms were chiefly manifested in the right arm, and from the headache, the stupor, the bruise on the left temple, I should have been justified in inferring that probably the front part of the brain was injured at the site of the bruise.   Had I opened the skull at that point, I should have found a perfectly normal brain and should have missed the clot.   The young man, therefore, would have died whether his skull was opened or not.

"In 1902 observe the difference.   As a result of the knowledge derived from experiments upon animals, which have located precisely the center for motion of the right arm on the left side of the brain near the top and a little in front of a vertical line drawn through the ear, and disregarding entirely the site of the headache and the bruise, I reached the conclusion that there had been a rupture of a blood-vessel within the head which had poured out a quantity of blood and that the situation of the clot should correspond to the 'arm center.'   The location of this arm center was far away (about three inches) from the location of the

bruise, and its position was fixed absolutely as a result of experiments upon animals, confirmed later by many operations on human beings, and also by postmortems.

" As soon as the skull was opened at this point, the clot was found, its thickest point being exactly over the arm center, and nine tablespoonfuls of blood were removed, with the result that the patient's life was saved. The blood had first been poured out over the 'leg center,' which is located higher up than that for the arm. This explained the long spasms in the right leg. The clot did not extend, however, further down than the arm center. This explained why the face was never convulsed, for the face center lies just below that for the arm. . . . . The patient made a complete recovery.''

THE POINT OF OPERATION.

The physician who undertakes to map out the site for the operation of trephining in epilepsy must possess not only a definite knowledge of the great variety of epileptic aura, but he must know the important facts of cerebral localization as well. These two things constitute the supreme guides at our command. If the convulsions are foreshadowed by numbness and twitching in the hand, or by convulsive movements in the leg or foot or side of the face, or by some disturbance of vision, hearing, taste, or smell, or by any distinct localizing symptoms, we must follow these initial manifestations back to their probable point of origin and be able to locate the area in the brain from which they came.

There is no other disease in which a thorough knowledge of cerebral localization is so important and none in which the guides to location are so clear and specific as furnished by the aura in epilepsy when fully studied and properly interpreted.

When cases are met with in which the localizing

symptoms do not correspond to the site of the injury, it is best to follow the medical indication, operating at the point which should give rise to such symptoms and not at the point of injury.

Traumatic epilepsy is often indefinite in its expression; that is, a local injury does not always produce a local fit; or again the fit may be confined to certain parts for months, and then, without known cause, become general. This tendency probably represents the worst phase of the affection; it means a more widespread implication of brain tissue in the process of degeneration, a lessened power of resistance under repeated attacks. If the patient is naturally prone to disease, this retrogression will appear all the quicker, so that the cardinal rule in every surgically treated case of epilepsy should be, *operate as soon after the receipt of the injury as possible: never let a second convulsion follow the first when due to injury, if it can be prevented by surgical intervention.*

Trephining in idiopathic epilepsy is sometimes done under the assumption that the convulsions are due to increased intracranial tension. Kocher, in Switzerland, and several American surgeons did this operation quite extensively a few years ago; at present it has almost fallen entirely into disuse.

It is far more rational to operate in idiopathic cases with localizing symptoms than in idiopathic cases without such symptoms, even though the results under the former up to this time have been disappointing.

A distinctly localizing symptom does not always mean a localized condition or disease susceptible of removal by the knife. The fact has repeatedly been made patent to us. The woman previously mentioned who had 519 seizures in forty-nine hours and a half, the initial contraction each time being confined to the first phalanx of the right thumb, upon whom an autopsy was performed at the end of that time, and whose

brain showed (under careful pathologic study) abso-
lutely no trace of local disease save a condition of
cell-vacuolation, present as a *result* and in no wise a
cause, is a typical case in point.

We could mention others in which the attacks con-
fined to one part of the body for years disappeared
temporarily upon the excision of the corresponding
brain center only to recur in the same site later on.
A girl of thirteen, an epileptic for six years, whose at-
tacks averaged from seven to ten daily, nearly all begin-
ning in the left hand, suffered excision of the left hand
center. All power was lost in the hand for several
weeks. Five days after the operation the attacks
recurred, but were general in character. With the
return of power in the left hand the attacks again began
in that member.

Repeated observations in cases like these strengthen
the belief that epilepsy is oftener due to widespread
cortical conditions or disease than to any circum-
scribed agent removable by the knife. When localiz-
ing symptoms occur in such cases, they indicate the
weakest point in the brain area diseased, the excision
of which necessarily fails to affect the larger fault.

The organic epilepsies arise from such causes as
tumor of the brain, including tubercle, syphiloma,
glioma, sarcoma, and others of less frequency such as
myxoma, fibroma, carcinoma, osteoma, lipoma, psam-
moma; in fact all forms of new growths found else-
where may cause convulsive phenomena by appearing
in the brain. Faults in the vascular system, hemor-
rhage, profuse or capillary, emboli, thrombi, and
aneurysms are also occasional causes.

It is often a matter of great difficulty to diagnosticate
brain tumors in epilepsy, either as a cause or as a
concurrent affection, for the reason that the two dis-
eases often produce almost identical symptoms.

The only class of epileptics in which the diagnosis

can be made with any degree of certainty is that in which the disease is recent, with the mind but little impaired, and with paroxysms far enough apart to give the patient opportunity for reaching his normal state between attacks.

Tumors of the brain in epilepsy must therefore be diagnosticated during the inter-paroxysmal period when the symptoms of tumor are not overshadowed by the symptoms of epilepsy, such as headache, paralysis, defective vision, and the like.

The proportion of operable cases of brain tumor is exceedingly small. According to White and Starr, only 2 per cent. of them justify surgical measures. Bergman and Kroenlein conclude that the indications for operation in such cases are very limited.

With the x-rays and with our advancing knowledge of cerebral localization, together with the advances of modern surgery, we may be led to a point where the prognosis under such conditions will be more favorable than it is at the present time. Certain it is that the most momentous fact in the entire domain of the surgical treatment of the disease lies in *the early recognition of the cause*, and in this the x-rays seem now to promise aid in some cases.

Peripheral Operations.—The causes of epileptiform convulsions that are often amenable to surgical treatment include numerous injuries and diseases in various parts of the periphery. Thus, when a trauma involving a nerve trunk has started the convulsions, we may try revulsives, then denudation of the nerve, stretching, or section if necessary. It is a good rule to resort to local measures in cases of this kind first, and in a majority of cases they will be found effective, provided the convulsions are not of too long standing.

Sometimes (Féré) insignificant irritations involving the terminal extremities of a nerve may be the cause of all the trouble, and their removal may effect a

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cure. The same is occasionally true in regard to foreign bodies in the ear or the nasal fossa. Nasopharyngeal polypi on removal have caused the disappearance of epileptiform crises. Féré speaks of the cure of *auricular epilepsy* (vertiginous attacks) by relieving the Eustachian tube of temporary obstruction by insufflations of air. The relief of stricture of the urethra, an inherent prepuce, and other conditions in connection with the reproductive organs which require surgical correction, sometimes check reflex convulsive phenomena.

The relief of eye-strain as a possible cure of epilepsy is fully discussed under the general treatment of the disease in the preceding chapter.

Mention may be made of such other surgical measures as *counter-irritation*, *ligature* of the *vertebral arteries*, and *resection* of the *cervical sympathetic*. Under the former a seton is usually put in the back of the neck. "That this does occasional good is testified by strong though ancient evidence" (Gowers). This writer, however, failed to secure any good results from its use. Accidental burns that cover large areas are said to produce results similar to those from the use of the seton. We fail to understand the value of counter-irritation as ordinarily applied in the treatment of epilepsy.

Alexander, of Liverpool, was apparently the first to recommend and try ligature of both cerebral arteries. At best it was an ill-conceived procedure and is no longer thought of.

Janesco * appears to have revived the plan of complete bilateral resection of the cervical sympathetic as a possible cure of epilepsy. He treated three patients in this way. Improvement followed in them all for the first five months, in none did convulsions recur in that time, while infrequent and transitory

* "Centralblatt für Chirurgie," Jan. 16, 1897.

attacks of vertigo appeared in but one.   Janesco concludes that total bilateral resection of the cervical sympathetic may be practised without secondary evil results, and that the operation is worthy of further trial, basing the belief on the demonstrated disturbance of the cerebral circulation during attacks.

Carl Beck * reports three cases in which he performed resection of the sympathetic ganglia of the neck, all being cases of "genuine epilepsy" of long standing.   A decrease in the frequency and severity of attacks occurred in all cases, but gradually the old condition returned.

Roswell Park performed this operation on three patients at the Craig Colony, and while sufficient time has not elapsed to note permanent results, improvement was noted in some respects immediately after.

RESULTS OF SURGICAL INTERVENTION GENERALLY.

It is exceedingly difficult to sum up the results of the surgical treatment of epilepsy for two reasons:

1. Those who report cases commonly base their conclusions on personal conceptions as to what constitutes genuine epilepsy and what does not.   This is bound to be inevitable in the absence of any fixed nomenclature based on a more scientific foundation than that of symptomatology.   One operator or reporter may regard epileptiform convulsions as essential epilepsy, while another may look upon some irregular form of the latter as being epileptiform in character.

2. The lack of any limit fixed by rule or common consent as to the period of time that should elapse after the operation and before results are announced, is a second fruitful cause for misleading and imperfect statistics.

A few years ago the tendency to report results almost immediately ("before the wound heals,"

* "Chicago Medical Recorder," April, 1899.

Bergman rather caustically remarks) was greater than now. Happily more conservatism is beginning to be manifested in this respect.

It would be unscientific to attempt to lay down any rule as to time for reporting results after operation, applicable to all cases alike. Recent convulsions in distinctly traumatic cases give definite results before cases classed as idiopathic and of long standing. The same principle, in fact, applies here that is so important throughout the whole field of the study of epilepsy and its treatment, namely: everything must be determined upon an individual basis—generalizations are too often misleading.

A third stumbling block that the surgical treatment of epilepsy has frequently to encounter is that of delay after the receipt of the injury before operation is undertaken. *The time to operate is at once*, before the seal of chronicity is set beyond the possibility of removal. The more marked the indications of heredity as a factor in causing the disease, the closer the operation should follow the trauma, for the disease in such cases very soon becomes ineradicable.

While the literature is replete with the results of surgical intervention in epilepsy, the following cases briefly cited from the Craig Colony records fairly illustrate the value of this form of treatment:

Case I.—A man aged twenty-nine years. Family history negative. Epilepsy began at seventeen years; supposed cause, malaria. Type, *grand mal*, idiopathic. Trephined in October, 1895, six years after the first seizure. Results, negative.

Case II.—A man aged thirty-one years. Father, tuberculous; family history otherwise negative. Epilepsy began at twelve years. Supposed cause, trauma to the head. Trephined over the right motor region in August, 1894, eleven years after the origin of the epilepsy. Results, negative.

Case III.—A young man aged twenty-two years.

Family history negative. Epilepsy began at fifteen years. No assigned cause. The patient grew rapidly worse from the outset, soon having as many as twenty-four attacks in a day. Type, *grand mal*. Trephined in October, 1897, three years after the first fit. Following the operation the attacks became fewer in frequency, but more severe in character. Trephined a second time at the Colony in April, 1900. The attacks were growing steadily worse. A portion of thickened and adherent dura was removed. Following the second operation, the patient showed decided improvement, due probably as much to careful dieting, nursing, and treatment in other respects, as to the operation *per se*. Results, great decrease in frequency and severity of attacks.

Case IV.—A man aged thirty-one years. Maternal grandfather, syphilitic and insane. Maternal aunt, insane and a suicide. One brother and one sister died in convulsions. Paternal relative, intemperate. Epilepsy began at seventeen years. Type, *grand mal*. Supposed cause, cranial trauma at seven years. Trephined in 1895, eight years after the onset of epilepsy. Results, negative.

Case V.—A man aged thirty-eight years. Maternal grandmother, epileptic. Assigned cause, heredity. Type, *grand mal*. Trephined over the left motor region in 1893, fifteen years after the onset of epilepsy. Result, slight temporary improvement.

Case VI.—A boy aged eight years. Family history negative. Epilepsy began at three years. Supposed cause, trauma to the head from a fall. First attack two months after the fall. Attacks increased in frequency so that at the age of five years the patient was having fifty a day. Two years after the first convulsion he was trephined over the right motor region. Following the operation he had no attacks for three years during the day, but occurred during the night just the same. Result, decrease in number of attacks.

Case VII.—A man aged thirty years. Mother, rheumatic; father, inebriate and died of tuberculosis. Epilepsy began at twenty-one years. Assigned cause, injury to the left side of head by being run over by a

wagon.   First convulsion two weeks after the accident.
Attacks at first, three or four daily.   Type, traumatic
grand mal.   Trephined one week after the first attack.
After the operation he had an average of twenty each
month.   Result, slight decrease in number of attacks.

Case VIII.—A young man aged nineteen years.
Family history negative.   Epilepsy began at eleven
years as the result of a blow on the head with a bale
stick.   First convulsion occurred half an hour after
the injury; the second a month later, after which they
occurred with increasing frequency.   Trephined in
1896, three years after the injury.   No benefit as a
result of the operation.   He was trephined a second
time at the Colony in April, 1900, this time over the
left motor region, this being the site of the injury.
Nothing of a pathologic nature was found.   Type,
grand mal.   Result, no improvement.

Case IX.—A man aged thirty years.   Family history
negative.   Epilepsy began at seven years; supposed
injury to the head at the third year.   Trephined in
February, 1899, sixteen years after the injury which
was supposed to have caused his epilepsy.   At first
all his attacks were psychic; later they changed to
grand mal and were not benefited by the operation.
Result, no improvement.

Case X.—A young man aged twenty years.   Mother
and maternal grandmother died of tuberculosis.
Epilepsy developed at twelve years.   Assigned cause,
penetrating wound of the skull by a nail when eight
years old.   First trephined over the left motor area
in 1899, four years after the injury.   Trephined again
at the Craig Colony in November, 1900, over the same
area.   Thickened dura removed and gold foil inserted.
Result, no improvement.

Case XI.—A man aged thirty-six years.   Father,
alcoholic.   Maternal uncle and aunt, insane.   Mother
and grandmother had organic heart disease.   Epilepsy
began at twenty-four years.   Supposed cause, trauma
to the head a year previously.   The patient claims to
have received a fracture of the skull at that time by
falling from a ladder.   First convulsion occurred twelve
hours after the accident.   Attacks after that occurred
every six weeks.   Eleven months later he was tre-

phined over the left frontal region in the Presbyterian Hospital, New York. Six weeks after the operation a convulsion occurred. Shortly afterward he was given large doses of bromid. Result, improvement; no attack for six months after the operation.

Case XII.—A boy aged eleven years. Mother, neurotic. Maternal grandmother had two strokes of paralysis. Epilepsy began at eight years, caused by falling from a shed a distance of eight feet, striking on the right side of head. Had a convulsion the same day of the accident. In April, 1900, two years after the injury, he was trephined over the right motor area at the site of the injury. Result, no improvement.

Case XIII.—A man aged thirty-one years. Family history unknown. Epilepsy began at nineteen years. Supposed cause, yellow fever, contracted in Brazil in 1891. The first convulsion occurred shortly after this. In 1893, two years after the beginning of epilepsy, he was trephined over the right frontal region. Result, no improvement; attacks more frequent after the operation than before.

Case XIV.—A young man aged twenty years. Family history negative. Epilepsy developed at the age of fifteen years. When five years old he was pushed off a wagon and injured his spine. First attack occurred a month later. A year after that he fell from an engine and was unconscious some time. In September, 1900, four years after the onset of his epilepsy, he was trephined. Result, no improvement.

Case XV.—A boy aged fifteen years. Family history negative. Epilepsy began at six years. Assigned cause, trauma from being struck on the head with a shovel. First attack occurred a month later, appearing three or four days after that. Nine years after the injury he was trephined over the site of the injury. Result, epilepsy worse after operation than before.

Case XVI.—A young man aged nineteen years. Family history negative. Supposed cause, trauma to the head by being struck by a train. First convulsion occurred eight years later, appearing about every ten days after that. Trephined four years after the receipt of the injury. Result, the disease was worse after the operation.

Case XVII.—A man aged twenty-nine years. Family history negative. Epilepsy began at the age of twenty-one years, after he had been thrown from a wagon, striking on the back of his head. Trephined two years later. Enjoyed freedom from attacks four months after that, when they recurred. After that he complained of severe pains in his head. Result, epilepsy not improved.

Case XVIII.—A woman aged twenty years. Family history negative. Epilepsy began at thirteen years. First convulsion occurred fifteen months after the patient fell, striking the right side of her head on ice. Trephining over left motor area in November, 1894. During the five months following the operation she had no seizures, then they recurred as formerly, and two years later were manifested at the rate of four or five a month. Result, some temporary but no permanent benefit.

Case XIX.—A woman aged twenty-seven years. Paternal great-grandfather and grandfather died insane. Father, alcoholic. Two maternal aunts, alcoholics. Maternal uncle died insane. Mother, epileptic since she was sixteen years old. The patient's epilepsy began at the eleventh year. Assigned cause, heredity. At twenty years she was trephined over the left motor area and had no attacks for a year after that. Then the attacks recurred as bad as ever. Result, marked temporary but no permanent improvement.

Case XX.—A man aged thirty-eight years. Family history negative. Epilepsy began at twenty-seven years. Assigned cause, injury to the head by being caught between two ice wagons. Trephined over the right Rolandic region eight years after the injury. Result, no improvement.

Case XXI.—A young man aged twenty-two years. Family history unknown, except that all (?) the paternal relatives were said to be intemperate. Epilepsy began at eleven years. Supposed cause, kicked by a horse. Trephined over the seat of the injury; dura found thickened. Result, no improvement.

Case XXII.—A girl aged eight years. Father, alcoholic. Epilepsy began at two years, following an infantile cerebral palsy. Trephined at four years. Skull very thick. Result, no improvement.

Case XXIII.—A woman aged thirty-eight years. Family history unknown. Epilepsy began at eight years, following a fall downstairs. A second injury was caused by a runaway horse. Convulsions followed the latter accident immediately. She was unconscious at the time, remaining so for three days. Trephined over the left motor area at the end of the third day. The convulsions continued with varying frequency until she was thirteen, when they entirely disappeared until she was thirty. Then they returned and persisted for eight years. On her admission to the Colony her uterus was found impervious and was removed with both ovaries. Result, three years after the operation she had suffered no further attacks; cure probable.

Case XXIV.—A girl aged nine years. Family history unknown. Epilepsy began at three years. No assigned cause. Three years later she was trephined over the left motor area. Result, no improvement.

Case XXV.—A man aged thirty-two years. Multiple sclerosis. Trephined by the family physician in June, 1901. Operation over left motor area. Result, no improvement in epileptiform symptoms due to his multiple sclerosis.

Case XXVI.—A young man aged twenty years. No occupation. Epilepsy of six years' duration. Right hemiplegia. Jacksonian type. Trephined by Dr. Gerster at the Mt. Sinai Hospital, in November, 1901. A piece of bone two inches and a half square was removed. Result, no improvement.

Case XXVII.—A man aged forty years. Epilepsy came on a year previously. Family history negative. Type of epilepsy, both *grand mal* and *petit mal*, occurring frequently. The right arm and left leg were most frequently affected. Trephined six months after being injured on the head by a falling stovepipe. Attacks recurred twelve days after operation. Result, no improvement.

Case XXVIII.—A boy aged ten years. Family history negative. Epilepsy of nine years' duration. Cerebral palsy; right hemiplegia. *Grand mal* attacks beginning on the right side of the face, right arm, and

leg. Trephined at the Colony in May, 1902. Result, no improvement in epilepsy.

Case XXIX.—A man aged twenty-nine years. *Grand mal* epilepsy for twenty-five years. Cause, typhoid fever. Paralysis of left arm. Trephined at the Massachusetts General Hospital seven years before admission to the Colony. Result, no improvement.

Case XXX.—A boy aged sixteen years. Epileptic since the second year. Cause unknown. Attacks both mild and severe, generally beginning in the left hand, extending up the arm and left side of the face and head, then to the left leg. Trephined at the Colony in June, 1902. No marked pathologic condition found. Result, no improvement.

Case XXXI.—A boy aged sixteen years. Received a fracture of the skull when seven years old; trephined soon afterward. Epilepsy came on two years after the injury. No heredity; no paralysis. Attacks average two or three a month. Result, no improvement.

Case XXXII.—A man aged twenty-six years. Epileptic since the fourteenth year. Cause, trauma to the head. Type, *grand mal*. Paralysis of the right arm and leg, and left side of the face. Trephined in Syracuse in 1899. Result, seizures more frequent after the operation.

Case XXXIII.—A young man aged twenty-one years. Epileptic since the seventeenth year. Cause, a blow on the head by a pitchfork. *Grand mal* convulsions. No paralysis. Trephined over the right motor area previous to admission to the Colony. Result, no improvement. The patient is an imbecile.

Case XXXIV.—A girl aged thirteen years. Epileptic since the sixth year. Epilepsy followed a severe enterocolitis attended with high temperature and convulsions. A condition of semispastic rigidity followed in the left hand, arm, and leg, lasting for some weeks. Her attacks occurred at the rate of from seven to ten a day, mostly at night. They usually first appeared in the left hand, and extended in definite order each time. All of the attacks were essentially motor; there was no sensory aura of any kind, and as soon as the fit was over the patient's mental condition was as clear as ever. She was never comatose afterward,

though she occasionally slept from exhaustion follow-
ing the severer attacks, sleep being preceded by a
few moments of waking state following the fit.   Type
of attacks, *grand mal* and *petit mal*.   Trephined at the
Colony in May, 1903, over the left motor area by Dr.
Roswell Park.   The left hand center was located by
electrical stimulation and completely excised.   Noth-
ing pathologic was found.   Result, slight improve-
ment.*

Twenty out of the thirty-four cases here cited were
due to trauma to the head, or to supposed trauma;
the rest were mostly idiopathic, some having a distinct
cause, while all had more or less persistent localizing
symptoms.   In six of them the operation was per-
formed at the Colony;   the rest, by skilled surgeons
elsewhere.

The average duration of the epilepsy before the
operation was approximately five years and a half,
being sixteen years in one case and three days in an-
other.   In no case (save one) was the result noted less
than eleven months after the operation; in the majority,
several years had intervened. . In twenty-one there
was no improvement in the disease, the attacks being
neither lessened in frequency nor severity.   In nine
the attacks were favorably modified to some extent.
In three the disease was much worse, this being
noticeably true in Case XIII, in which the cause was
yellow fever, and the operation over the right frontal
region was performed two years after the first con-
vulsion.

In one case only was there apparent recovery.   Here
trephining followed trauma to the head at eight years,
but failed to check the epilepsy, the convulsions con-
tinuing five years longer, when they spontaneously
disappeared to recur at thirty.   So the apparent cure

* It is entirely too early to speak of permanent results in this case.   Less
than three months had elapsed between the date of the operation and the
date on which this was written.

in this case must be ascribed to the results of abdominal section and not to the earlier operation on the brain.

Twenty-eight of the thirty-four cases were males. Twelve of these had good family histories, ten were unfavorable in this respect, while in six the history could not be ascertained. Of the six women, three had good family histories, two had not, while in one it was unknown.

It is worthy of note that none of the patients died as the result of the operation.

Similar treatment of idiocy and imbecility (craniectomy) is attended with a mortality rate of from 20 to 25 per cent.

As a rule, epileptics tolerate surgical operations well.*

SEIZURE RECORDS IN SIX CASES TREPHINED AT THE CRAIG COLONY.

Case I.—Operated April, 1900.			*Case II.—Operated April, 1900.*		
	1900.	1901.		1900.	1901.
January	559	1	January	3	4
February	136	3	February	1	2
March	131	3	March	7	1
April	205	1	April	13	3
May	14	2	May	1	3
June	0	0	June	11	2
July	3	2	July	1	3
August	0	4	August	1	1
September	0	7	September	6	6
October	0	7	October	1	8
November	0	1	November	5	3
December	3	0	December	4	4
Total	1051	31	Total	54	40

* From the "Ninth Annual Report of the Craig Colony," p. 44. "Sixty-four operations of importance were performed in the Peterson Hospital during the past year; three were capital operations on the brain; five were double ovariotomies; others for appendicitis, resection of elbow, etc. From a surgical standpoint, the results in all cases were uniformly good, all operations, without exception, being followed by rapid and satisfactory convalescence."

Case III.—Operated Nov. 25, 1900.   *Case IV.—Operated May 7, 1902.*

	1900.	1901.		1902.	1903.
January	12	4	January	0	5
February	14	5	February	0	11
March	31	6	March	0	3
April	13	2	April	2	3
May	19	3	May	4	0
June	16	6	June	2	0
July	0	4	July	5	0
August	0	11	August	1	0
September	5	14	September	2	0
October	13	10	October	2	0
November	2	12	November	4	0
December	0	9	December	0	0
Total	125	86	Total	22	22

We have no accurate information regarding the seizures in Case IV before admission, and are unable to compare the number of attacks before operation with those occurring afterward.

Case V.—Operated June 7, 1902.

	1902.	1903.
January	10	25
February	13	13
March	208	75
April	5	2
May	6	0
June	5	0
July	13	0
August	7	0
September	2	0
October	7	0
November	10	0
December	18	0

The large number of seizures in Case V in March, 1902, was due to status epilepticus.

Case VI.—Operated May 26, 1903.

April 26	3	May 26 Operation.	
" 27	8	June 4	2
" 28	12	" 5	0
" 29	7	" 6	0
" 30	2	" 7	1
May 1	1	" 8	2

May			June		
"	2	7	June	9	3
"	3	9	".	10	7
"	4	4	"	11	7
"	5	1	"	12	6
"	6	4	"	13	10
"	7	4	"	14	5
"	8	3	"	15	3
"	9	7	"	16	2
"	10	1	"	17	7
"	11	3	"	18	5
"	12	1	"	19	6
"	13	2	"	20	5
"	14	2	"	21	3
"	15	3	"	22	3
"	16	3	"	23	2
"	17	3	"	24	0
"	18	3	"	25	3
"	19	4	"	26	6
"	20	2			—
"	21	2	Total after		88
"	22	1			
"	23	2			
"	24	4			
"	25	2			
"	26	1			

Total before 111

The attacks in Case VI are given for a period of twenty-eight days before and twenty-eight days after the operation only. This suffices to show the feature most pronounced after surgical intervention, namely, the temporary benefit the patient often derives that seldom becomes lasting.

Briefly noted, the results in the foregoing six cases were as follows: In Case I there were 1031 attacks during the four months preceding the operation. During the four months following it there were 17 only, while during twenty months following it there were 51 attacks.

In Case II there were 24 attacks during the four months preceding the operation, and fourteen during the four months following it. This ratio of decrease kept about the same afterward.

In Case III there were 125 attacks during the eleven months preceding the operation and 66 during the eleven months succeeding it.

In Case IV the number before the operation could not be ascertained. During the first twelve months following it there were 45.

In Case V there were 242 attacks during the five months preceding the operation, and 54 during the five months succeeding it; while during the ten months following it there were 193.

In Case VI there were 111 attacks during the twenty-eight days before the operation, and 88 during the same period after it.

It will thus be seen that while absolute cure was not obtained in any of the thirty-four cases reported, a marked improvement followed in about a fourth of them. Five of the six patients trephined at the Colony had been operated on previously.

Matthiolus collected 258 cases of Jacksonian epilepsy, the most favorable of all epilepsies for surgical treatment, in which the patients were subjected to craniectomy. "Some 20 per cent. were reported as cured, though only ten of the entire number had been followed for over three years, and only eighteen of them for over one year. Of the others, 15 per cent. were reported improved; 56 per cent. unimproved; 13 per cent. died."*

Brown collected thirty cases due to trauma, in which thirteen were reported cured, nine improved, and eight unimproved.

Ferrier reported twenty-one cases of partial epilepsy, with twelve recoveries, six ameliorations and negative results in three; adding, "it must be remembered that such observations are often published before six months have elapsed."

* Roswell Park, "American Medicine," Vol. IV, Nov. 21, 1902.

Starr* gives the surgical results in twenty-nine cases as follows: "Cured ten, improved six, not improved eleven, died two."

It must be noted, however, that these conclusions were drawn some years ago (1892) when results were reported sooner after the operation than is generally done at the present time. Most of them were noted at three, four, six, and eight months after operation.

It is apparent to the unbiased student of epilepsy long familiar with the disease in its many aspects, that conservative surgery has a place of value in its treatment.

It is, at the same time, fully as apparent that much of the surgical treatment of epilepsy of the past was done under less knowledge of the etiology and pathology of epilepsy than need be the case to-day. Our advancing conception of its pathogenesis seems likely now to restrict surgical work in its treatment still further than is the case to-day.

Neither the epileptologist nor the surgeon can at this time better advise as to the probable cause of capital surgical intervention for the possible relief of idiopathic epilepsy than to say: It is one of the most obstinate diseases of the brain the physician is called upon to treat. After all other treatment, fairly tried, has failed, a surgical operation may offer relief; far less often it promises cure.

The majority of those operated on experience no benefit, while about a fourth are aided some. Under ordinary circumstances and strict antiseptic precautions, little danger is to be feared from the operation itself.

TECHNIQUE OF TREPHINING IN EPILEPSY.

Preparation of the Patient.—The patient's head should be entirely shaved the day before the operation;

* "Brain Surgery," 1893, pages 55 to 67.

the scalp washed with soap and water and thoroughly scrubbed with a nail brush, after which it should be washed with ether, and finally with a 1 : 1000 solution of bichlorid of mercury.   An aseptic gauze dressing wet with a 1 : 2000 solution of mercuric bichlorid is then put on and held in place by a properly applied bandage which is left in position until the operation.

Immediately preceding the operation a quarter of a grain of morphin sulphate or two drachms of the fluid extract of ergot may be given for the purpose of contracting the cerebral blood-vessels and diminishing hemorrhage.   Suspension of the bromids for some days before the operation has been advocated, but it is best to continue them sparingly for fear of status.

Immediately before beginning the anesthetic, the dressing on the head may be removed and the point of attack carefully mapped out.   To do this successfully, a knowledge of cerebral localization and of the surgical anatomy of the skull and brain is essential. The center to be attacked must be determined by the localizing symptoms exhibited during the convulsions, and the point on the skull under which such center lies marked for identification.

In epilepsy following traumatism to the skull, where evidences of injury are superficially apparent, it may not be necessary to resort to accurate measurements to locate the point at which to trephine.   When the evidences of trauma and the localizing symptoms do not point to the same portion of the brain, the skull should be opened at the point indicated by the localizing symptoms.   The medical rather than the surgical indications in such cases are more likely to guide us to the seat of irritation.

In cases of traumatic epilepsy without localizing symptoms, when the trauma involves a so-called silent portion of the cortex, the external signs of injury may be the only guide.   In the majority of cases the

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centers most frequently sought lie about the fissure of Rolando, so that the accurate location of the fissure is of great importance. The location of the fissure of Sylvius is not so important, but in some cases with aphasic or auditory symptoms it must be mapped out.

The fissure of Rolando is located by drawing a line on the scalp from the root of the nose to the external occipital protuberance. A point thirteen millimeters posterior to the middle of this line lies above the upper end of the fissure which runs downward and forward from this point at an angle of 67°. This fissure is about 3⅜ inches long and its lower third is slightly more vertical in direction than its upper two-thirds.
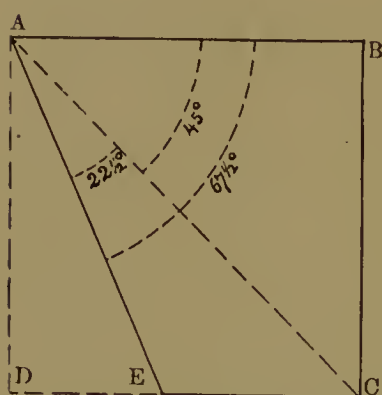
Fig. 16.—Chiene's method of fixing position of Rolandic fissure.

Many methods have been suggested for marking the location of this fissure on the scalp. One of the simplest, and which for practical purposes is sufficient, is that of Chiene, who folded a square of paper diagonally. (See diagram.) The angle B A C is 45°. By again folding the paper and bisecting the angle C A D, the angle E A D is formed, which is 22½°. The angle E A B is 67½°. This angle is accurate enough for practical purposes, and if the side of the paper A B is now placed on the scalp coinciding with the line joining the root of the nose and the external occipital protuberance, the point A being placed thirteen millimeters posterior to the middle of this line and the side A B running forward from that point, the side of the paper A E will indicate the position of the fissure, which can be marked on the scalp with iodin or an anilin pencil. A few touches of the cautery will serve to make the line indelible and recognizable after the final washing of the scalp.

1ST FRONTAL CONVOLUTION
2ND FRONTAL CONV.
3RD FRONT. OR BROCA'S CONV.
Head
Neck
Writing Memories
Eye Movements
ANTERIOR
Upp-
Lips-Lo
Ton a
Vocal Cords
Speech Memories
Thr t
H
1ST TEMPO
Word
2ND TEMP
3RD TEM
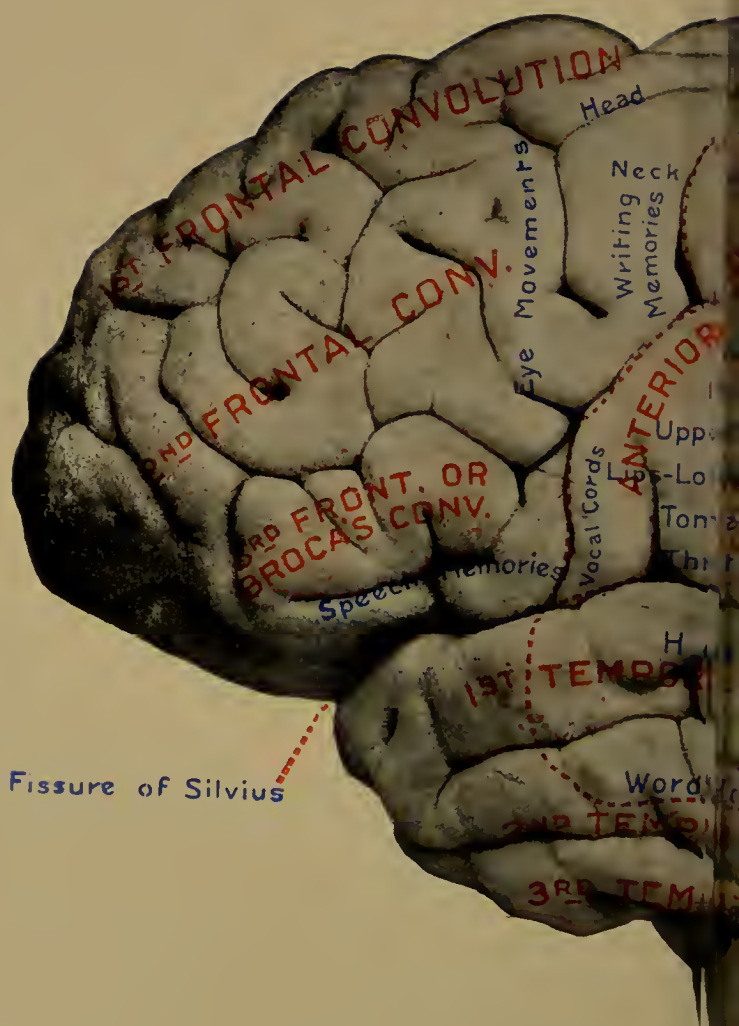
Fissure of Silvius

Cortical centers of the brain,

Fissure of Rolando

Foot
Leg · Toes
Knee
Shoulder
Elbow
Wrist
Muscle Memories
Word Vision
Parieto-occip
Fissure.

POSTERIOR CENTRAL CONV.

SUPRA MARGINAL C.

ANGULAR CONV.

1ST OCCIP.

2ND OCCIP.

Visual
Sensation
Memories

OCCIP.

3RD OCCIP.

CONV.

nories
L CONV.
AL CONV.
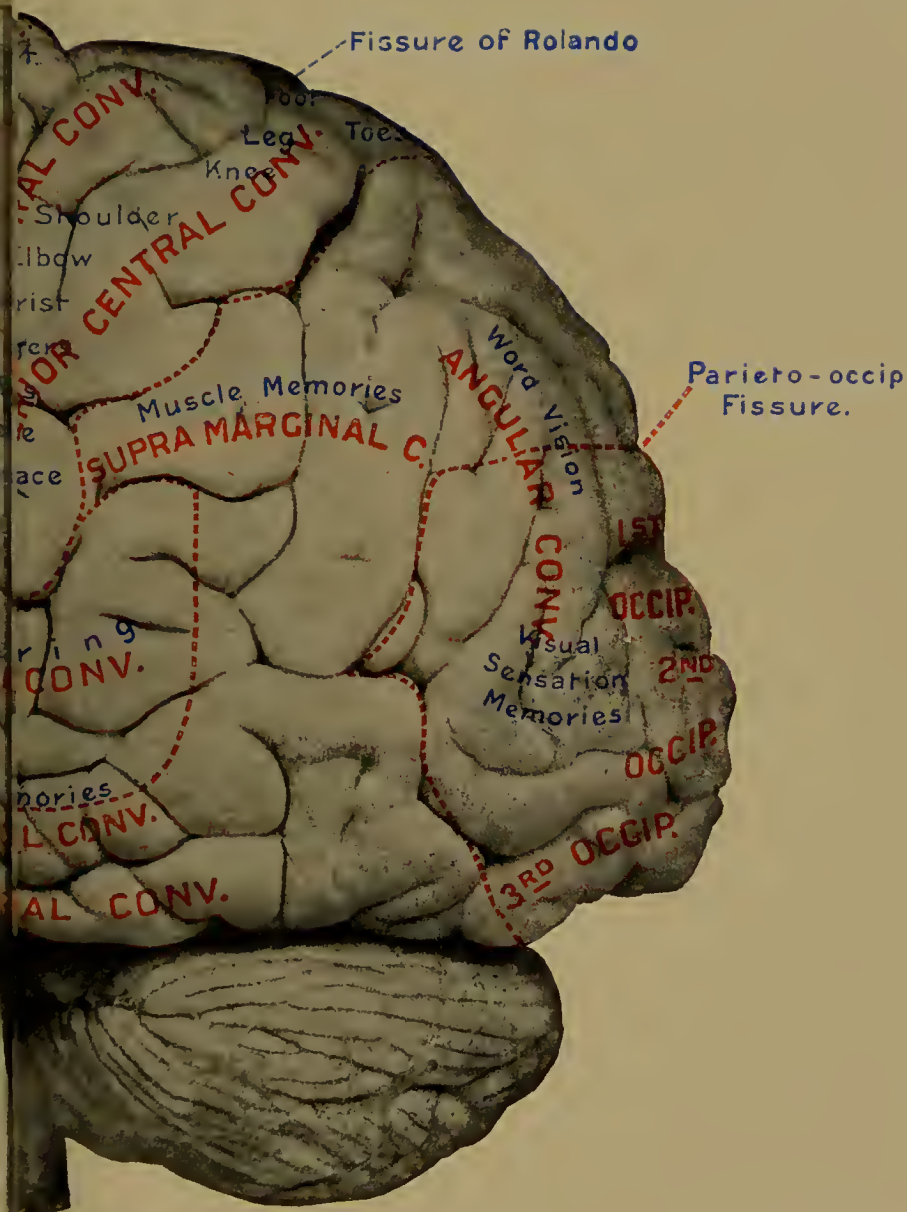
surface (modified from Dalton).

The fissure of Sylvius is located by drawing a line directly backward from the external angular process for an inch and a quarter and then upward for a quarter of an inch. A line is then drawn from this last point to a point three-quarters of an inch below the most prominent portion of the parietal eminence. This line lies over the horizontal portion of the fissure. The point of junction between the horizontal and ascending branches of this fissure lies about two inches behind and slightly above the external angular process of the frontal bone, the ascending branch running upward from this point for three-quarters of an inch behind the coronal suture.

The location of the desired fissure having been marked on the scalp, the exact point at which the center pin of the trephine is to be placed must also be determined and marked. The anesthetic is next given, the scalp again washed with a 1 : 1000 solution of bichlorid, and the head placed in a good light on a firm support. After the patient is anesthetized, the skull should be marked at the point to be trephined by puncturing the scalp at the point with the center pin of a trephine or other instrument and nicking the bone. The scalp may also be punctured at two points along the line indicating the direction of the fissure, the bone being marked deeply enough to be recognizable after the scalp flap is reflected. Unless the bone be marked in this manner, it is difficult to decide at what point the trephine should be applied.

While these directions are useful in determining the point of attack, the skilled surgeon, especially in Jacksonian cases, often dispenses with them, making the incision simply over the motor area, and removing sufficient bone to permit extensive exploration of the cortex by means of electric stimulation. The seat of disease can often be better detected in this way than by the most careful external measurements. The cortex need not be exposed to apply the faradic cur-

rent.   Stimulation can as well be obtained by applying
the electrodes to the dura before it is opened.*

Instruments.—The following instruments should
be in readiness: two scalpels, a periosteal elevator, a
trephine, a rongeur, a pair of small blunt scissors, a
tenaculum, a pair of dressing forceps, a probe, a grooved
director, some fine curved needles, a small spatula, a
number of artery forceps, catgut and silk ligatures.
A Paquelin cautery, a faradic battery, and a small
electrode (like Keen's) may be required; and Horsley's
wax—composed of wax seven parts, carbolic acid one
part, and oil two parts—may be useful during the
operation.

Operation.—The incision should be made base down-
ward, semi-circular or horseshoe in shape, with due
regard for the blood-supply of the flap and subsequent
drainage of the wound, should this be necessary.
The incision should be ample in extent to give room
for removing the necessary bone without further
enlargement of the scalp opening, or having the edge
of the bone opening encroach too near to the line of
incision in the scalp.   The scalp incision can be
made with one or two sweeps of the knife, prompt
pressure with gauze sponges being used to control the
hemorrhage as the incision is made.   Bleeding points
at the flap edges are caught up with artery forceps
and the hemorrhage promptly controlled.   These
forceps can be left in place for some time.

The flap is raised, the periosteum reflected, and the
bone laid bare.   The center pin of the trephine is then
placed at the spot previously marked on the bone and
the instrument applied vertically to the plane of the
portion of the skull attacked.   After the bone is
grooved sufficiently, the center pin is withdrawn and

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*The choice of an anesthetic is important; if chloroform be used the
head should be slightly raised to lessen the danger of hemorrhage. [See
"Anesthesia," under Abdominal Section.]

Plate 23.
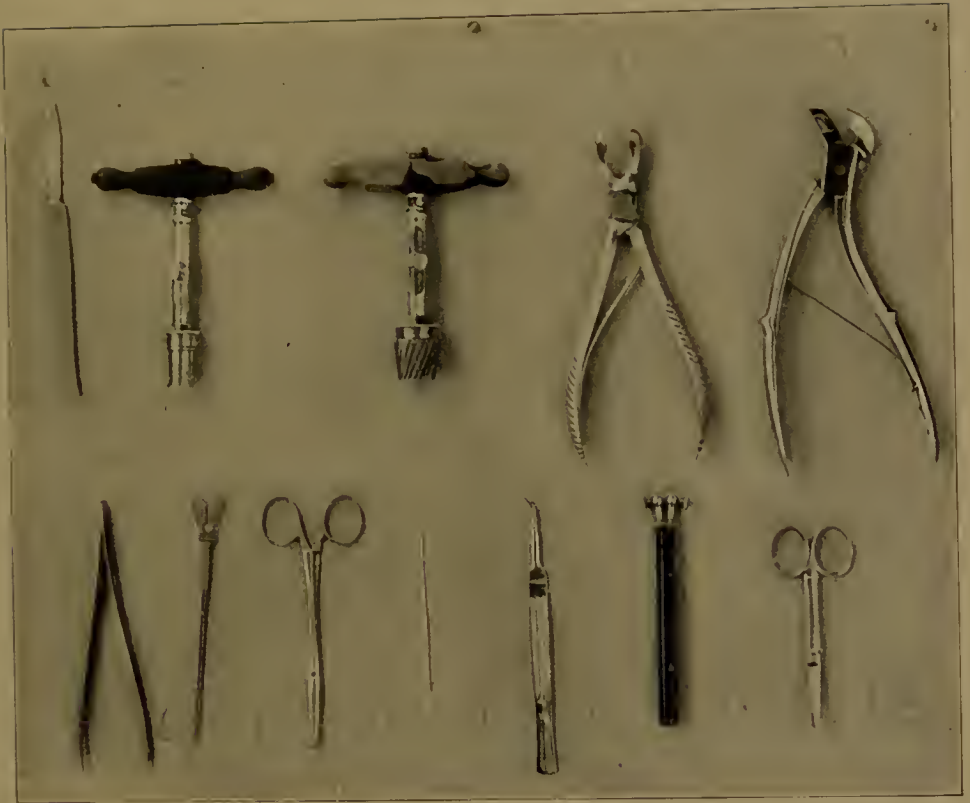
INSTRUMENTS USED IN TREPHINING FOR EPILEPSY.

1, Scalpel; 2, 3, large and small trephines; 4, 5, bone-cutting forceps; 6, dressing forceps; 7, grooved director; 8, forcipressure; 9, probe; 10, Horsley's dural separator; 11, Keen's electrode; 12, scissors.

the trephining continued carefully, the operator remembering that the skull may differ considerably in thickness at different points in the circumference of the trephine opening. The trephine must be withdrawn at times to free it from bone detritus and to permit the exploration of the opening by means of a probe to see if the cranium has been cut through at any point.

When the diploë has been entered, the resistance to the trephine will be lessened and blood will appear in the opening. When the inner table has been cut through, the button of bone will be movable, and it may be removed either by rocking the trephine or by prying it up with an elevator. The trephine used should by preference be large, or two or more openings may be made with a smaller instrument and the intermediate bone cut away with rongeur forceps. Whatever the method, there should be no hesitancy in removing sufficient bone to give ample room for exploration and the space necessary for doing any operation the conditions may demand. It is always a mistake to attempt to operate through a small opening.

After the button of bone is removed, it is well to separate any adhesions between the bone and dura about the opening by means of Horsley's dural separator, or a probe bent at an angle, before enlarging the opening with the rongeur. In cutting away the bone, if the field of operation is in the vicinity of any of the sinuses, care must be taken not to injure these structures. Fortunately, operations for the relief of epilepsy are not frequently done near a sinus and the danger from this source is not great. The hemorrhage from the bone is often considerable, but can usually be controlled by pressure with hot compresses, by plugging the bleeding point with a few strands of catgut, gauze, or even with a piece of match or a

toothpick; also by the use of Horsley's wax or the actual cautery. If these do not suffice, the two tables of the skull may be forcibly crushed together at the bleeding point with heavy forceps. Should a sinus be wounded, the hemorrhage may be controlled by plugging with gauze.

When the dura has been sufficiently exposed, it should be opened by lifting it away from the under-lying structures with a tenaculum and incising it with the point of a scalpel. The incision can then be carried around parallel with the edge of the bone opening and about a quarter of an inch away from it, making a flap in the dura with its base downward. If any of the large branches of the middle meningeal artery are encountered, a ligature of catgut can be passed under them by using a fine curved needle and the vessel ligated before the dura is opened.

After the dura is opened, the further course of the operation will depend on the conditions found. Adhesions between the dura and the underlying membranes about the margin of the opening should be broken up with a probe properly bent. This may be done before the dural flap is made by introducing the probe through the first incision made in the dura and sweeping it around carefully. On opening the dura the appearance of the brain may give valuable aid in diagnosis. At times, the pia may be so edematous as to obscure the outlines of the convolutions. In such cases it will be necessary to incise the pia to allow the escape of the fluid and permit an inspection of the surface of the brain.

Bulging of the brain into the trephine opening is an evidence of increased intracranial tension which may be due to the presence of a cyst, tumor, abscess, or internal hydrocephalus. Absence of pulsation in the part of the brain presenting is also evidence of the same conditions. Discoloration of the surface may

be an indication of tumor beneath the cortex. Palpation also gives valuable information. Undue firmness is indicative of tumor; fluctuation, of an abscess or cyst. The brain can be depressed carefully and the finger introduced under the dura and swept around the margins of the opening and the cortex explored for some distance.

Sufficient information can usually be obtained by inspection and palpation, but if necessary the brain may be explored with the hypodermic needle or by incision. The latter methods should not be resorted to indiscriminately and will not often be necessary in operations for the relief of epilepsy. If the brain be explored by puncture or incision, the entrance should be made over the summit of a convolution and the direction of the incision should be vertical to the portion of brain attacked. Scars in the dura or cortex resulting from old lacerations should be sought for. Depressed bone and bone splinters demand removal, and adhesions should be broken up wherever found. The remains of old clots can be removed by gentle wiping with gauze with the assistance of irrigation with warm sterile water.

Cysts are best treated by excision whenever practicable; where they cannot be dissected out, the superficial portion may be removed, the interior emptied, cauterized, and drained. Tumors, when encapsulated, can be enucleated with scissors and spatula. When not encapsulated, they must be removed by dissection through the healthy tissue. Scars in the dura should be excised, while cortical scars should be carefully dissected out with scalpel and scissors.

Scars of the cortex to be removed can be outlined with the scalpel and cut away with scissors. In doing this there is less danger of wounding other important cortical centers by incisions made in the anteroposterior direction than by incisions made from above

downward. Such incisions need seldom extend deeper than the gray matter, unless the condition demanding removal extends more deeply. In certain cases none of these pathologic conditions may be apparent, and excision of the cortical center controlling that part of the body in which the convulsive movements have their origin may be considered.

The center is located by faradization of the cortex with a pair of small electrodes, using a mild current. The electrodes must be sterilized before using them and the handles wrapped in sterile gauze when in use. Antiseptic solutions must not come in contact with the electrodes when in use, as they interfere with electric action. When the center is located, it is removed in the manner described above for excising scars of the cortex. If benefit is to follow such procedure, the removal must be thorough and the entire center excised.

In the course of such operation, hemorrhage either from the pia or the brain substance may be troublesome. If from the pia, it may be controlled by pressure with hot compresses or by the cautery. It may be possible at times to tie off with fine catgut the bleeding portion of the pia *en masse*. If from the brain substance, it may be controlled by pressure with gauze wrung out of hot sterile water. A 1:40 solution of antipyrin recommended by Park, or a 1:100 solution of cocain recommended by Keen, is a useful styptic. A 1:1000 solution of adrenalin chlorid may be applied, and is usually effective in lessening this form of hemorrhage. If the bleeding is excessive and persistent, the wound may be tamponed with gauze.

After the source of irritation has been removed as thoroughly as possible, the wound is closed. In every case the dura should be closed, if possible; a continuous suture of fine catgut, applied by means of a small curved needle, being used for this purpose. In cases

where cysts or tumors have been removed and the resulting cavities are packed with gauze, or where drainage of any kind is used, it will, of course, be impossible to close the dura completely. In such cases an opening may be left at the most dependent portion of the dural flap, through which the drainage material may pass.

On account of the danger of adhesions forming after such operations, the introduction of some material to prevent their formation is necessary. While no perfectly satisfactory method has been devised for attaining this end, the insertion of gold foil is, perhaps, as satisfactory as any. If it is used, a piece large enough to completely cover the exposed cortex and extend under the margins of the dural opening should be carefully inserted before the dura is sutured. Suture of the dura may be interfered with by protrusion of the brain through the opening. This may be overcome by pressing it back with small spatulas inserted under the dura while the suturing is in progress.

It is, perhaps, better not to attempt replacing the bone in these cases. The scalp is closed with silk sutures. Gauze drainage may be used, if necessary, or a few strands of silkworm gut may suffice. In cases where it is important to insure patency of the scalp wound and efficient drainage, the gauze drainage material may be allowed to separate the edges of the scalp wound for some distance at the dependent portion of the opening. Silk sutures are introduced at regular intervals along the scalp edges at this part of the wound, but left untied until the drainage is removed on the second or third day. An abundant aseptic dressing is finally applied. Where no drainage is used, and there are not symptoms to the contrary, this dressing may be left in place for a week or ten days, when the wound will probably be completely healed. When drainage of a cyst or brain cavity has been

effected, it will be necessary to dress the wound more frequently and to gradually remove the drainage in accordance with the demands of the case. In any case, excessive discharge resulting in extensive staining of the dressing calls for its removal and reapplication. Symptoms of pressure from retained secretions demand the removal of the dressing and the examination of the wound. In such cases it may be necessary to remove one or two sutures to permit the escape of these secretions. An increase in temperature with evidences of infection calls for the immediate examination of the wound. As a rule, the sutures may be removed eight or ten days after the operation.*

ABDOMINAL SECTION.

Indications for Abdominal Section for the Possible Relief of Epilepsy.—In this connection the acute inflammatory and malignant diseases that affect the reproductive organs in women and which may require operation irrespective of the presence of epilepsy, are not considered.

The pathologic conditions within the abdomen, which may be a factor in causing or increasing the frequency of epileptic seizures, are generally those affecting the ovaries and uterus, the condition having persisted a sufficient length of time to set up reflex nervous symptoms.

In some cases of severe dysmenorrhea the ovaries will be found enlarged and with numerous cysts beneath a thickened capsule. This may be a real excitant to determine the epileptic seizures at the menstrual period, and it constitutes one of the so-called menstrual epilepsies. The removal of such

* *References.*—"Hospital Records at Craig Colony." Esmarch and Kowaltzig, "Surgical Technique." Bryant, "Operative Surgery." Dennis, "System of Surgery." "American Text-book of Surgery." Deaver, "Surgical Anatomy." Senn, "Practical Surgery." Tillmanns "Text-book of Surgery." Starr, "Brain Surgery."

ovaries often lessens the frequency and severity of the epileptic seizures.

When the ovaries are normal and the epileptic seizures are more frequent at the menstrual period, they should not be removed. A dysmenorrhea may be relieved, but the mental and nervous symptoms which follow the artificial production of the menopause will be likely to increase the tendency to mental failure so frequently a result of epilepsy as it is.

Anterior and posterior displacements of the uterus, with the ovaries and tubes bound down by firm adhesions, also constitute conditions productive of reflex symptoms in various parts of the body, and may act unfavorably upon the patient's epilepsy.

In rare cases the uterus may be a retention cyst, the pain and reflex symptoms due to the retained menstrual flow becoming so intense in predisposed subjects at the menstrual period as to produce distinct epileptic phenomena—convulsions that closely simulate the true disease, and which, in selected cases, may eventually pass into it, so far at least as the final effects are concerned. Mention has previously been made of a woman at the Colony who had this condition, the relief of which through removal of the uterus and ovaries checked all further attacks.

It behooves us to make careful inquiry into the possible influence of perverted functions on the part of the reproductive organs in any case of epilepsy among women occurring after puberty and before the menopause.

In some cases in which epilepsy comes on before puberty, or arises after it, independent of any disease of the ovaries or uterus, operation may be justified on the score of lessening the frequency and severity of epileptic attacks that are often prone to great augmentation at the monthly flow. But it is not an operation under any circumstances for indiscriminate

adoption. It requires careful selection, and then it must be regarded in most cases as a part of the treatment only.

Preparation of the Patient for Abdominal Section.— As most of the cases requiring operation are of a chronic nature, the time selected for the operation should be a period most remote from the attacks. Usually this is not difficult when the attacks are prone to occur in series about the menstrual epoch.

The patient should be thoroughly prepared for the operation by observing the following points for a week beforehand: The urine should be carefully examined a number of times. If the patient is not taking bromid, it is generally best to give some form of the drug in moderate amount during the operating period. The diet should be restricted to easily digested foods, giving nothing but liquids for a day before the operation, and no food at all for six hours before the anesthetic. A saline cathartic should be given twenty-four hours before; an enema the night before and on the morning of the operating day.

Following the warm bath on the day preceding the operation, the skin of the abdomen is prepared by scrubbing with potash soap and warm water; all hair is shaved from the abdomen and pubes; a poultice of green soap is applied over the abdomen and retained for three or four hours; the abdomen is scrubbed again with soap and sterile gauze sponges and all soap is removed with sterilized water; it is then scrubbed with alcohol and ether and washed with a 1 : 1000 solution of bichlorid of mercury; a pad of gauze and absorbent cotton saturated with a 1 : 4000 bichlorid solution is then applied and kept on until the time of the operation.

A vaginal douche with hot soapsuds, followed by a

1 : 5000 bichlorid solution is given on the morning of the operation. If any discharge is present, this treatment is given for several days previous to the operation. The cleaning of the vagina may be postponed until the patient is under the anesthetic, when it is scrubbed with green soap on cotton sponges, but it should be done in all cases, especially when the uterus is to be operated upon.

Preparation of the Operator and Assistants.—A part of the outside clothing should be removed and a sterile gown or duck suit worn by the operator and assistants. A rubber apron may be worn under the gown to protect the clothing, and a sterile linen cap should cover the hair of the head.

The hands and forearms should be thoroughly scrubbed with potash soap and hot water, using a sterile brush. Then immerse the hands for two minutes in 95 per cent. alcohol and thoroughly clean the nails and fingers. Finally the hands and forearms are immersed in a 1 : 1000 solution of mercury bichlorid for a few minutes, and then rinsed in sterile water.

Everything that comes in contact with the field of operation must be sterile.

Instruments are best sterilized by boiling for thirty minutes in a one-per-cent. solution of carbonate of soda. If they are put in the water after it has begun to boil, the danger of rusting will be lessened.

Silk for sutures and ligatures may be sterilized by steam or by boiling in a one-per-cent. soda solution.

Silkworm gut is sterilized by superheated steam or by boiling in plain water.

Silver wire may be boiled in a solution of carbonate of soda, or it may be heated direct in the flame of an alcohol lamp.

Catgut is rendered sterile by boiling after first treating it with formalin, according to the method of Hofmeister. The catgut is wound on glass rods or plates

and immersed in a two- to four-per-cent. solution of formalin for from twelve to forty-eight hours and then washed in running water to remove the formalin. It is next boiled for thirty minutes and then preserved in alcohol containing five per cent. of glycerin and one-tenth of one per cent. of mercury bichlorid.

Dressings, towels, etc., are sterilized by steam under pressure.

Plenty of sterilized normal salt solution, hot and cold, should be ready.

Anesthesia.—Ether is the safer anesthesia in most cases. By starting with chloroform and changing when narcosis is well under way to ether, some of the unpleasant effects of the latter can be avoided. Before giving the anesthetic, the bladder should be emptied, using a catheter if necessary. A hypodermic injection of morphin sulphate, $\frac{1}{6}$ to $\frac{1}{4}$ of a grain, and atropin, $\frac{1}{150}$ to $\frac{1}{75}$ of a grain, given just before the operation, will lessen nervous excitability and render the administration of the anesthetic safer and easier.

When the patient is well under the anesthetic the abdomen is prepared for the operation by again scrubbing with sterile soap and water, washing with a bichlorid solution and then with sterile normal salt solution. A piece of sterile gauze is placed over the area to be operated upon and the surrounding parts covered with towels wrung out of an antiseptic solution, such as 1 : 1000 bichlorid of mercury. The temperature of the room should be about 80° Fahrenheit.

The Operation.—The patient is placed in the Trendelenburg posture, with the hips elevated from fifteen to thirty degrees.

The incision is usually made in the median line between the umbilicus and symphysis pubis. A short incision is made at first, cutting the layers down to the subperitoneal fat. This is picked up with forceps and incised, exposing the peritoneum. All bleeding por-

tions should be secured and tied before opening the peritoneum. The peritoneum is then picked up with forceps and, being sure that it is free from the viscera, a small opening is made with the knife or scissors. A grooved director is inserted and the opening enlarged sufficiently to introduce the index and middle fingers of the left hand, and the incision then enlarged to the desired extent by cutting with a probe-pointed bistoury. Use broad retractors and a good light to obtain efficient observation of the field of operation. The abdomen is now ready to carry out the operation indicated.

After the completion of the operation and before closing the incision in the abdomen, the hips are lowered from the Trendelenburg posture to observe if hemostasis is complete. All bleeding points within the abdomen must be ligated and all oozing arrested. Remove all blood and clots by gently wiping with sterile gauze sponges.

Irrigation is unnecessary except in infected cases in which it is done with normal salt solution at a temperature of 112° F.

Closure of the Incision.—In closing the abdomen it is desirable to use a method which will give a firm union and which will *stand the strain of an epileptic seizure* should one occur during the healing process or afterward.

The best method for this is the one which unites the corresponding layers separately in the relation in which they existed before the operation. By this method the peritoneum is closed with a continuous suture of fine catgut, all air being expelled from the abdomen before tying the suture. The wound is now cleaned with hydrogen peroxid. The edges of the cut fascia or muscles are brought together and held by mattress sutures of silver wire or silkworm gut placed about two inches apart. The spaces between these

are accurately united with interrupted catgut sutures. The subcutaneous fat and the skin is closed with continuous catgut sutures.

The line of the incision is now disinfected with peroxid of hydrogen, dried with sterile compresses, dusted with aristol, and covered with gauze compresses held firmly in place by adhesive strips. Over all this a large pad of gauze and absorbent cotton is held in place by an abdominal binder which is accurately fitted and fastened with pins. The lower edge of the binder is prevented from slipping up by perineal straps on each side.

The dressings are removed about the tenth or twelfth day in uncomplicated cases, while the patient should be kept in bed at least four weeks after the operation.

An abdominal supporter should be worn for several months after the patient leaves her bed, for fear the strain of an epileptic seizure might weaken the scar.

Drug Treatment after the Operation.—It is important to keep the patient quiet after the operation and to prevent, if possible, any epileptic seizures during the repair of the wound.

The bromids which were given before the operation, should be continued now in slightly increasing doses, and *in sufficient amount to control the attacks.* Following the operation they may be given by enema until the stomach regains its balance.

After the wound is firmly united the bromids may be reduced, but should not be entirely cut off for several months.

Results of the Operation.—The results of the operation, so far as essential epilepsy is concerned, will be disappointing in the majority of cases. In proportion to the degree that diseased abdominal conditions influenced the attacks, the better will be the results. If, in any case, such irritation was the sole cause of

the epilepsy, the correction of this may entirely relieve the disease. Unfortunately the causes of essential epilepsy are very rarely found where surgical intervention can correct it, and although the patient may improve as a result of relieving an irritation within the abdomen, the epilepsy still exists, with one less irritable point to modify its character and frequency.

Hysterectomy with Removal of the Ovaries and Tubes.—An incision from four to eight inches in length is made in the median line between the umbilicus and the symphysis pubis. Slight adhesions are broken up with the fingers; the denser ones, by careful dissection with the knife or scissors. The dense adhesions are best broken up by working from below, beginning on the side least adherent, after tying the ovarian and uterine arteries.

The uterus is lifted forward and to one side, putting the opposite broad ligament on the stretch, and the ovarian vessels of this side are ligated at the outer end of the broad ligament. A second ligature. or a clamp is placed about these vessels near the uterus and the vessels divided between the ligatures.

The round ligament of the same side is next divided after tying two ligatures near the uterus and cutting between these.

An incision is now made from one round ligament to the other by cutting along the line where the peritoneum is reflected from the anterior surface of the uterus to the bladder.

The anterior surface of the uterus below this line of incision is stripped of its peritoneum by pushing it down with sponges held in artery forceps, until the cervix is well exposed.

The uterine vessels of this side are next ligated close to the cervix and cut above the ligature, the uterine end being held with forceps. The cervix is cut through just above the vaginal junction, and as soon as the

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cervical canal is cut a plug of cotton or gauze is inserted in it.

The cervix is completely cut through and the uterus pulled to the opposite side, exposing the uterine vessels which have not been divided. These are clamped with artery forceps and divided above the clamp. Next, the ovarian vessels of this side are clamped and cut and the uterus and its appendages completely removed.

The uterine and ovarian vessels which are held by clamps and all bleeding points within the wound should be securely ligated.

The cervical canal is disinfected with peroxid of hydrogen, and the stump of the cervix is closed over the canal by catgut sutures uniting the anterior and posterior edges over the top of the canal.

The cut margins of the peritoneum surrounding the wound are next brought together and united with a continuous suture of catgut, thus covering the entire wound area with the peritoneum.

All blood and clots must be removed from the peritoneum by wiping with gauze sponges, and the incision in the abdomen closed.

Salpingo-oöphorectomy.—An incision from two to three inches long is made in the median line, the lower end of the incision reaching about two inches above the symphysis pubis.

The ovary and tube are drawn out of the incision by means of the index and middle fingers.

A silk ligature is passed through the outer portion of the broad ligament below the ovarian vessels and tied over the top of the infundibulo-pelvic ligament, external to the fimbriated extremity of the tube. Another silk ligature passes through the uterine end of the broad ligament to secure the uterine vessels and is tied over the top of the broad ligament close to the uterus. The utero-ovarian ligament is next ligated.

The entire ovary and tube are removed by cutting about half an inch from the ligatures.

As an additional safeguard against hemorrhage, the cut ends of the vessels in the pedicles may be drawn out with artery forceps and a separate catgut ligature applied to each one, or an additional ligature may be tied around the pedicle.

After all hemorrhage has been controlled the abdominal incision is closed.

RESULTS IN NINE CASES OF OÖPHORECTOMY.*

Case I.—A. B., age twenty-three years. Family history negative. Epilepsy at nineteen years; type, *petit mal.* Frequent attacks of major hysteria prior to operation. No epileptic attacks for fourteen months before operation. Right ovary hypertrophied and cystic; left, hypertrophied. Both tubes and ovaries removed. Result, surgically good. No attacks of any kind from the time of operation, February 2, 1902, until her discharge, November 14, 1902, a period of nearly nine months. Marked improvement in general physical condition and in disposition.

Case II.—D. S., age twenty-one years. Father alcoholic. Epilepsy at thirteen years; type, *grand mal.* Frequency of attacks before operation, ten in sixteen months. Operation on February 26, 1902. Old pelvic peritonitis. Both ovaries adherent; left could not be removed; right, hypertrophied and cystic; removed. Result, surgically good. She had twelve attacks in sixteen months following operation. Gain in weight and marked improvement in general health; no change in epilepsy.

Case III.—T. C. A., age nineteen years. Paternal grandfather alcoholic, cousin epileptic. Assigned cause of epilepsy, diphtheria. Type, *grand mal.* Frequency before operation, two attacks in twelve months. Operation on April 25, 1902. Both ovaries enlarged and cystic; both removed. Result, surgically good. She had six attacks in twelve months following the operation. General condition greatly improved.

* From the "Craig Colony Hospital Records."

Case IV.—L. B., age twenty-two years. Sister hysterical. Assigned cause of epilepsy, "ovarian irritation"; type, *grand mal*. Relative frequency of attacks before operation, sixty-nine in four months. Operation on September 20, 1902. Chronic pelvic peritonitis. Both ovaries hypertrophied; both tubes and ovaries removed. Result, surgically good. She had sixty-six attacks in ten months following the operation. Attacks much diminished in frequency.

Case V.—C. B., age thirty-one years. Mother had puerperal insanity. Assigned cause of patient's epilepsy, onset of menstruation at fifteen years; type, *grand mal* and *petit mal*. Frequency before operation, thirteen attacks in nine months. Operation on September 30, 1902. Both ovaries enlarged. Result, improvement in general condition. She had four attacks in seven months. The patient passed from observation at the end of that time.

Case VI.—M. F., age twenty-eight years. Mother epileptic; sister insane and also epileptic. Assigned cause, onset of menstruation at thirteen years; type, *grand mal;* frequency of attacks before operation, one or two each month. Operation on December 9, 1902. Left ovary enlarged. Both tubes and ovaries removed. Result, no change in epilepsy. She has had one attack each month since the operation. Some physical improvement.

Case VII.—This case is not included, as the patient died of shock following the operation.

Case VIII.—N. J., age twenty-nine years. Family history negative. No assigned cause of epilepsy; onset at twelve years; type, *grand mal*. Frequency of attacks before operation, five in four months. Operation on March 9, 1903. Old pelvic peritonitis. Peritoneum greatly thickened. Ovaries adherent; right ovary enlarged; left, atrophied and not removed. Result, surgically good. She had eight attacks in four months after the operation. No material change.

Case IX.—Age twenty-four years. Family history negative. No assigned cause; type, *grand mal;* onset at twelve years. Frequency of attacks before operation, none for five months. Operation on May 6, 1903. Result, surgically good. Unsatisfactory two

months later so far as attacks are concerned, the patient having had two within that time.

Omitting the seventh case, in which death followed the operation, the average length of time that elapsed after the operation and before results were noted, was nine months and nineteen days. Results: The attacks were reduced in frequency in Cases I and IV. The general physical condition was improved in Cases I, II, III, V, and VI, without visible improvement in the epilepsy. In Case VIII there was no change worthy of note four months after operation. In Case IX there was an increase in the number of seizures after the operation.

As a general rule—one subject to modification in especial cases—operations on epileptic women for the removal of the reproductive organs may be done if the attacks came on about puberty in the first instance, and occurred thereafter in close conjunction with the menstrual epoch, either singly or in small series, as is often the case; and they are apt to give most satisfactory results where hereditary causes are eliminated, and where the disease after years of action has failed to appreciably impair the faculties of the mind.

CHAPTER XVI.

THE PSYCHOLOGIC ASPECTS OF EPILEPSY.

Paroxysmal Mental States.   Psychic Epilepsy.   Automatism.   Pre- and Post-paroxysmal Insanity.   Transitory Ill-humor.   Emotional Irritability. Impulsiveness. Moral Anergia. Feeble-mindedness. Imbecility. Idiocy. Dementia. Manic-depressive States.   Degrees of Mental Impairment and Responsibility in Each. Genius and Epilepsy. Moral Decadence. Epilepsy and Crime.

At the outset we may lay down certain almost axiomatic facts pertaining to the effects of epilepsy on the mind, in the main as follows:

1. Every true epileptic convulsion destroys or impairs the integrity of the mental faculties to some extent.

2. Such effects cannot be measured by the degree of motor or psychic violence that accompanies the attack.

There are convulsions in which mental activity is completely effaced—all responsibility in abeyance during the progress of the attack—yet in which there is not the least perceptible trace of muscular commotion in any part of the body.

This is especially true of psychic epilepsy, which constitutes one of the most perplexing and interesting problems the medical jurist can encounter.

3. There is also a form of post-paroxysmal automatism very similar in character to psychic epilepsy, characterized by acts apparently performed under the direction of conscious volition, seemingly logical and in perfect sequence, fulfilling a definite purpose; yet all through the performance of them the patient is no more responsible than if he were an inanimate machine. In this automatic state all the functions

of the body, even that oftentimes of speech, act in a coördinate and natural manner, although the field of conscious life is a perfect blank.

It is difficult to fully realize the true character of this condition. My own skepticism of it was considerable until I had witnessed it in so many instances in which I felt absolutely assured that the patient was under the influence of a morbid psychologic entity over which he had not the slightest control.

4. In addition to the states of mental vacuity just described, we class the effects of epilepsy on the mind in other ways in point of time as temporary, prolonged, or permanent. The temporary effects include transitory states of disturbance that appear with the convulsion, are inseparable from it, because they are essentially a part of it, and disappear with it.

The prolonged effects include forms of disturbance that precede or follow the convulsion, lasting variable lengths of time, and assuming many different types, while permanent forms of alienation include all varieties of mental unsoundness, from simple loss of memory to complete epileptic idiocy, imbecility, and dementia.

Before we go further, and in order that we may possess as clear an impression as possible of the whole subject in a concrete form, let me restate the matter in this way.

Paroxysmal Mental States Due to Epilepsy.

1. *Psychic epilepsy.* A complete morbid entity in itself and wholly destructive of responsibility so long as it is present.

2. *Epileptic automatism.* A condition of mental vacuity coexisting with natural bodily activity. This usually follows severe attacks, though it may be induced by those of milder form.

3. *Pre- and post-paroxysmal mental disturbances.* These are usually in the form of the most violent and

destructive mania, lasting anywhere from a few minutes up to days or weeks, and in rare cases even longer.

4. *Paroxysmal or epileptic mania.* This is always destructive and dangerous in character, and in which the mental disturbance coincides with the fit. In cases in which it appears to be a substitute for the fit, it is known as the psychic epileptic equivalent.

Inter-Paroxysmal Mental States Due to Epilepsy.

1. Transitory ill-humor and simple loss of memory for recent events; in other cases, for events regardless of the time of their occurrence. To these we must add as pronounced adjuncts of the epileptic's mental peculiarities: emotional irritability, impulsiveness, moral anergia, and incapacity for any form of valuable productive occupation dependent upon the initiative in conception and consecutive activity.

2. Slight clouding or dulling of the intellect, as a whole, which often becomes more pronounced just before the fit.

3. Feeble-mindedness.

4. Imbecility.

5. Idiocy.

6. Epileptic dementia.

7. Acute confusional insanity characterized by delusions, hallucinations, and illusions. The latter form, under either condition, is quite rare. It is also rare in epilepsy to meet with acute states of depression characterized by painful delusions or by suicidal inclination or attempt.

We may readily group all the psychoses that come under the seventh heading under the broadly generic term *manic-depressive insanity*, which includes the usually recoverable forms of mania, simple and recurrent, melancholia simple and recurrent, and circular insanity, the latter being less curable.

While we seldom meet with circular insanity (the *folie circulaire* of the French) due to epilepsy or existing

coincident with it, we do find such cases occasionally. Two of the kind have come under my observation, both ending in permanent dementia.

PSYCHIC EPILEPSY AND EPILEPTIC AUTOMATISM.

The effect of psychic epilepsy in cases in which the disease exists for years, vary greatly. It has generally been assumed that this was intellectually the most destructive of all the epilepsies, but this is not true in all cases.

Much depends upon the etiology, but more upon the age at which it develops and the stamina of the patient. I have observed that when it appears early in life in individuals whose family history is good and whose stamina is of superior kind, such attacks may occur daily for years without appreciably affecting mental integrity. I recall one case in particular, that of a girl of seven years whose seizures appeared from forty to sixty times daily for five years, when they began to show a marked decrease, the child developing naturally in every respect. Least of all was there any blunting or retardation of her intellectual development.

In other cases in which the disease is delayed in its establishment until adult life, and when it is preceded by vicious and unwholesome living, especially alcoholic excesses, it begins almost immediately to mar the refinement of the psychic life, and in two or three years destroys it almost completely. Fortunately, psychic epilepsy is comparatively rare. There is no doubt, however, that it sometimes exists for years in an unrecognized form.

It may be either diurnal or nocturnal in its expression, and is especially apt to escape notice when it occurs only at night, being in some instances mistaken for somnambulism.

"We are apt to lose sight of the fact that the slightest

seizures are just the very cases in which consciousness
is prone to be most impaired or involved, and in which
a seizure is most likely to be overlooked by the friends
or even the patient himself, and thus it happens that a
paralysis of the central hierarchy of the nervous system
may so withdraw control over lower centers as to
issue in wild excitement, although the epileptic seizure
was so slight as to be scarcely, if at all, appreciable
to the onlooker'' (Bevan Lewis).

Epileptic Automatism.—It is quite remarkable how
somnambulistic individuals unconsciously guard them-
selves from harm while in such a state. In walking
about, they go with their eyes closed or opened, or
partly opened, but it is plain to see that when the eye
is open it does not see.

Such individuals appear to act with deliberate pur-
pose, avoiding places that are dangerous and never
striking objects that produce injury. The instinct
of self-preservation seems as active at such times as
under normal conditions. I am personally familiar
with several patients with whom a rational conversa-
tion may be carried on while they are in an automatic
epileptic state, but I have never witnessed a case in
which any knowledge of the conversation could be
recalled by the patient, provided the automatism was
complete, after the automatic period had passed away.
It has been my observation that the acts, mannerisms,
peculiarities, habits of vocation, and the like, shown
by the individual in his normal state, can be carried
over into and most perfectly repeated in the fullest
detail in the automatic state, *but that it is impossible
for the automaton to carry the memory of such acts out
of this state into the conscious state beyond.* I have re-
peatedly tested this point and have never found an
instance in which it did not hold true.

As a rule, the psychic or automatic epileptic, during
the access of the attack, is not in a normally receptive

Plate 24.

E. C., who had 40 to 60 mild seizures a day for seven years, yet whose mental development proceeded naturally. At the age of fourteen years (her epilepsy began at seven years) she was having a single attack only once in three to four months. This case is presented to show that psychic epilepsy does not always destroy the mind.

condition, so far as special sense impressions are concerned. If they are spoken to, it must be done loudly for them to hear and understand, and it is often necessary to accompany spoken commands with a gesture.

Many suffer from partial deafness at such times, but can readily interpret the meaning of a gesture, the visual apparatus being apparently unimplicated in the attack.

The automatic state is usually one of motor tranquillity, though it may be the reverse. There are cases in which marked combativeness is aroused by any attempt to exercise control over, or to aid, such a person. A young man afflicted in this way was riding on an elevated train in New York when the guard noticed that he appeared ill, and grasped his arm to assist him. The patient immediately struck at him in great fury and a violent struggle ensued, ending in the arrest of the patient and his detention by the police, until he regained consciousness, when he established his claim as an epileptic and was permitted to go.

A male epileptic had a psychic seizure of fifteen minutes' duration. He answered questions intelligently, though I had to shout them loudly to make him understand. He sauntered about the room looking at various objects, and was perfectly calm and rational to all outward appearances, until I tried to lead him over to the light to examine his eyes. He jumped back, assumed a threatening attitude, and made as though he would strike me. A little coaxing finally induced him to yield, though it was easy to see that he was suspicious of harm. His face wore a puzzled, apprehensive, pained expression of a type that I have never seen duplicated save in other epileptics during the aura and the early part of some convulsions. Fear, being one of the most primitive instincts, appears to be retained and unconsciously expressed by the

epileptic while in this state. It seems to be the auto-matic operation of the law of self-preservation.

One redeeming feature of psychic epilepsy in its most profound form, that is, when a single attack lasts for hours or days, is, that such attacks are separated by wide intervals. When the attacks are rapid in suc-cession and consist merely of momentary "blanks," they may occur scores of times in a day.

The medicolegal aspects of this type of epilepsy depend, so far as responsibility is concerned, upon our ability to determine the existence of the automatic state at a given moment. This may be difficult to do beyond reasonable doubt, though if we can prove that the person is a sufferer from epilepsy at the time, or ever had it in any form, we can always create a reason-able belief that the patient may have acted while in a seizure, without any intent whatever, and under con-ditions that should free him from responsibility.

The evidence of the presence of epilepsy, already presented in detail in a previous chapter, needs to be carefully studied in order to arrive at a just conclusion in medicolegal cases. If it can be proved beyond reasonable doubt that the individual has epilepsy, the question of responsibility is not difficult after that. We may not be able, it is true, to say positively that he was under the influence of a seizure at the moment an overt act was committed, while, on the other hand, we are equally as unable to prove that a seizure was not present. Psychic convulsions defy all ordinary methods of detection. They can readily be noted, however, by one trained in the observance of their expression.

One of the strongest points in clearing the patient of responsibility for crime is the utter absence of motive. In other cases the *apparent* motive may be out of all proportion to the brutality and degree of the crime committed under the explosive violence of

Plate 25.

P. McS., photographed while in a state of epileptic automatism.   The patient was unconscious at the moment the picture was made, remaining so half an hour afterward—this state following a severe grand mal attack. Criminal acts by epileptics while in this state are not uncommon.

an epileptic attack that, itself, may have been incited by a slight provocation. In such cases it may be difficult to determine the degree of responsibility. But it is always safe to argue that every epileptic suffers from defective inhibition. The power of self-control, under the faintest causes that challenge it, is gone, a fact that in itself places the epileptic in the irresponsible class.

Psychic epileptics may commit all manner of crimes: theft, arson, rape, assaults, homicides. They are not infrequently pyromaniacs entirely without reason or impelled by the flimsiest motives.

An epileptic boy of twelve years, of fair intelligence and previous good conduct, was given some stockings to wash that belonged to a fellow patient. The task displeased him, so he took them into the attic and set them on fire, the timely discovery of which prevented the loss of the building. He could give no reason for the act, and expressed regret over its occurrence. His conduct later was exemplary.

W. E. P., age forty-four years on admission. Epileptic since his eighteenth year. He was a clergyman by profession, and was accustomed to having from eight to ten attacks a month, both light and severe. He began to wander about the Colony collecting trash and starting fires in scores of places on the same day. He would burn up small sticks, straw, pieces of paper, and such débris wherever he found it. His whim was ignored for awhile, but later it was necessary to place him under restraint. He would never give any reason for doing this, but it seemed clear that he was acting under some morbid impulse. He was of a paranoiac type, constantly depressed, and refused ever to say that his health was good, for fear "God would punish him with a seizure for boasting."

People who suddenly disappear without known cause or purpose for indefinite periods and are finally

heard from in some remote locality, unable to explain how they happen to be there, should be suspected of being victims of this form of epilepsy.

The case of the commercial traveler previously mentioned illustrates this. In this instance the man went twenty-eight days in a subconscious state, performing the most complex acts, visiting different cities, traveling over many railroads, taking orders for goods, writing business letters complete in every particular, and making full notes daily in his diary. During the eighteen months I have known him he has had but one seizure. of this kind, though his attacks of *petit mal* are quite frequent and he is often automatic after them, doing various things for half an hour or so that he has no knowledge of later.

PRE- AND POST-PAROXYSMAL EPILEPTIC INSANITY.

Of the two conditions, pre- and post-paroxysmal epileptic insanity, the former is by far the more frequent. When it occurs, it may develop just before the fit, appearing suddenly in great violence, or it may begin days before in an insidious, accumulative way, the first changes affecting the disposition, which, usually placid and even, becomes irritable in spite of all efforts at self-control. Evidences of irritability increase. The patient becomes loquacious, finds fault generally, magnifies trifles into matters of great moment, is "touchy," suspicious, discredits the motives of all around him, is unable to sleep well, complains of disturbing dreams, suffers from headache, appears feverish and in a state of subdued excitement, finally acquiring pronounced falsification of special sense perceptions. These take the widest possible range, and constitute the aura of an approaching attack.

Some have delusions of persecution, others illusions, while hallucinations of sight also occur usually just

before the fit. These are mostly of a terrifying nature, though in rare cases they may be ecstatic.

Pronounced morbid depression just before the fit is rare, the mental state being more often that of dominant, confused ideation; the line of thought, as indicated by the speech, rapidly changing, the patient speaking in a quick, explosive way that gains in impulse as it proceeds, until an inarticulate jargon is the result.

Again, the flow of incoherent ideas may be less tumultuous, though more irrational in character, the patient talking the veriest nonsense in the most casual and self-contained way. Others express fixed insane ideas in an insistent way, without evidence of exaltation or depression.

A man of thirty-five years, whose infrequent attacks nearly always left him with sensory aphasia, calmly asserted for weeks that all the blood had been extracted from his body. Finally I stuck a pin rather forcibly into his finger one day, when he was surprised to see the blood flow. He did not mention his lack of blood again.

Another male patient of twenty-two years, of the finest physique, whose epilepsy followed too much riotous living, fancied he was being "played on" every night by electric waves sent through the walls of his room for the purpose. He suffered from the idea acutely just before his seizures, after which it was less insistent. In two months it left him, and his attacks ceased for several months, only to recur when he returned to the old improper way of living.

As a rule, disordered mental states that precede the convulsive periods subside completely with the coma stage that follows the fit—the acme of the often long-drawn-out epileptic state. Some patients fail altogether to remember incidents that happened during the pre-convulsive period of disturbance, which should always be held to include the period of irresponsibility.

PAROXYSMAL EPILEPTIC MANIA.

Some *grand mal* attacks are accompanied by the
greatest psycho-motor violence that it is possible to
conceive of. Such violence finds no parallel in intensity
in any other form of insanity. The patient is in a
state of the wildest frenzy (epileptic furor), rushing
blindly about in irresponsible rage, striking and assault-
ing every one he happens to come in contact with.
The more muscular the patient, the greater the possible
damage. Such epileptics seem to develop superhuman
strength. It has, in fact, been shown that the degree
of muscular power an epileptic may put forth during
a seizure is far in excess of that he can exert in his
normal state.

I have demonstrated this by placing the dynamom-
eter in the patient's grasp during serial and status
attacks. Patients of ordinary physique, men and
women, often show the most astounding strength
during the fit, requiring five or six strong attendants
to hold them.

These excessive frenzy states are generally brief,
rarely lasting more than a few minutes up to half an
hour, though they may be rapidly repeated, as I saw
in the case of a young mechanic who could only be
cared for with reasonable safety for days at such
times by covering the floor and walls of his small room
with mattresses. Such patients are always liable to
injury, both from their own violent movements, and,
in rare instances, accidentally through the well-meant
efforts of others in trying to restrain them.

Complete exhaustion usually follows such outbreaks,
often severe enough to keep the patient in bed for
days.   ·

It is impossible to determine the proportion of
epileptics who develop paroxysmal mania, but it may
appear in almost any type of the disease, and suc-

cessive attacks in the same patient may only be hours, or again years, apart.

We come now to the more *permanent effects* of the disease on the mind, the inter-paroxysmal states, which will be discussed briefly in regular order, beginning with the milder and more common varieties first.

TRANSITORY PERIODIC IRRITABILITY.

Through daily intimate contact with several hundred epileptics for a number of years, the writer has had abundant opportunity for observing what he has previously spoken of as the *temperamental* effects of the disease. It is scarcely a morbid entity, unless we call sudden unprovoked anger, marked irritability, sullenness, or ill-humor morbid entities also. At any rate, periodic ill-humor must be looked upon as often constituting a distinct forerunner of the convulsive attack as well as being a result of it.

Among the scores of cases I have been privileged to see almost daily for years, I have learned to detect with almost unfailing certainty—through noting temperamental changes alone the moment the patient enters the room and begins to speak—the approach of a convulsion a few hours or even days in advance of the convulsive period. An almost imperceptible change in personality has been wrought. The patient is querulous, fussy, fault-finding, nothing goes right; trifles that ordinarily produce no effect on him now completely engage his attention. His friends ignore him, his family is indifferent to his needs and his condition, his fellow patients are no longer congenial, their attacks disturb him, he cannot endure their jocose remarks, distorting them into expressions of ridicule. Finally, these ideas may persist in their growth, looming up larger and larger on the horizon of a morbidly heated mind, until they pass into qualified delusions, all being dependent upon the subtle,

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pernicious, autocratic influence of the approaching attack, and all completely disappearing, as if by magic, after the attack is over. In some cases, as we have stated, these ill-humor periods begin a day or so only before the fit, in others they come on weeks before, while in still other rare instances they come and go, without the occurrence of a seizure, being, as it were, a long-drawn-out, silently discharging seizure—*a fit without a climax*.

It is difficult to estimate accurately the proportion of epileptics who manifest to some extent these temperamental obliquities dependent upon the seizure periods, but it is entirely within reason to place it as high as from 75 per cent. to 80 per cent. or more.

Memory.—Of all the mental faculties the memory in epilepsy is the first to suffer, though we cannot say that it does so permanently in every case. Much depends upon the frequency, type, and severity of the attack—more upon the type than anything else. In motor epilepsy the attacks may be repeated weekly or daily for years, with scarcely any appreciable loss of memory. In other cases of greater psychic involvement with corresponding loss of motor disorder, the memory is affected early and in a marked degree, while in epileptic dementia, idiocy, and imbecility it is substantially completely destroyed.

I have repeatedly observed that a single seizure destroyed the memory of a thing an individual was especially charged not to forget. A skilled mechanic was sent to a neighboring village on an errand. Halfway there he had a mild seizure that left him automatic for a few moments. When consciousness was restored he was totally unable to recall the purpose of his errand, and had not done so an hour later when he was reminded of it. In the meantime, he appeared perfectly rational in every respect.

An epileptic carpenter was instructed to make a

simple piece of furniture from a drawing made in his presence at eight o'clock in the evening.   He studied the drawing with great care at the time.   Early the following morning he had a seizure, and on being questioned later, had forgotten the proposed work as completely as though it had never been mentioned to him. Finally the drawing was produced, when he at once exclaimed, " Ah! that's it; I remember it all now!"

The fact that isolated seizures destroy recent memory impressions makes it difficult to teach epileptics under purely intellectual systems.   A maximum of reiteration is required often with a minimum of results.

In determining the disintegrating effects of epilepsy on the memory, we must guard against mistaking the effects of the bromids in this respect for those of the disease.   It was almost a matter of daily occurrence, a few years ago, to admit patients to the Craig Colony whose mental bromism was so pronounced as to resemble acute dementia.

When memory impairment becomes fixed, a continuous dulling or clouding of the intellect is observed. The patient habitually forgets, loses all conception of time, becomes incapable of performing duties exacting in execution as to time, and demands constant urging and stimulation to endeavor.

The establishment of this condition foreshadows the beginning of **feeble-mindedness,** a state into which fully 50 per cent. of all epileptics sooner or later fall.   It is not so much a condition of helplessness as one in which the individual does better under some direction and control than without it.   With the approach of the paroxysmal period, this condition is heightened in intensity, to become again less marked as such period recedes.

The next grade in mentality below that of feeble-mindedness reached by many epileptics is **imbecility,** a condition less frequent than the former.

The imbecile is usually unable to receive many impressions, or to grasp and make use of daily experiences.   Individual and insignificant elements make up the sum of their experience.   According to Buccolo, thought with them is retarded—a more difficult proposition to prove than is the fact that it is scanty and along the most elemental lines.   Judgment is almost wholly lacking.   When present, it is defective and necessarily rarely based on experience, though it presupposes the presence of memory, which is feebly possessed, except in individual cases of imbecility, even by those of the higher grades.   ·

Consciousness is generally but little impaired, while memory for all but the most recent events is lacking. Occasionally trifling incidents are permanently remembered, while notable ones are forgotten.

The imbecile's knowledge of life is small; his world contracted and narrow; and he measures its influences and events largely by the manner in which they impress his personality.   The ego is magnified.   He lacks stamina or reserve force of any kind, and under extraordinary conditions he is wholly incapable of acting with salutary discretion.

The state of imbecility is often accentuated by epileptic crises and may be complicated with some show of violence.   Not infrequently it antedates the epilepsy, occupying to some extent the position of cause, though more often it follows it.   Epileptic imbeciles, without exception, are mentally incompetent under the medical test of responsibility.   Some, however, are shrewd enough to utilize their little knowledge in a manner to give their acts and assertions so fair a semblance of sanity and accuracy that they may be accepted as true by the layman or under legal definitions of responsibility.   It is to be regretted that they are not always judged from a medical standpoint alone.

Plate 26.

Characteristic types of epileptic imbeciles; the condition of imbecility, as a rule, preceding the epilepsy, the latter aggravating the former. Note hemorrhagic extravasation in the temporal canthus of the left eye of the patient on the right. Conjunctival hemorrhages are not uncommon after severe grand mal attacks.

Epileptic idiocy is the lowest mental state the epileptic can acquire, save that of *epileptic dementia.* In either case there is virtually a total loss of the faculty of thought.

This condition may be congenital or acquired, and, like epileptic imbecility, may stand as a cause of the epilepsy in part, or as the result of the convulsions. Its causes, as shown by the results of autopsies, include chronic encephalitis, diffuse or circumscribed; diffuse syphilitic disease of the blood-vessels; arrest of vascular development in the cortex; irregularity in the two hemispheres; inequality in the peripheral cortical layer on the two sides; defect of the third frontal convolution and the Island of Reil; meningo-encephalitis, with thickening and adherence of the pia and brain, such as may occur after forceps and trauma; cephal-hematoma internum; spontaneous hemorrhages; embolism from heart disease; thrombosis from cholera infantum, followed by destruction of cortical cells and atrophy of the cortex; macrocephalus and microcephalus. Any of these causes acting in conjunction with the disastrous effects of severe and prolonged epileptic seizures suffice to produce epileptic idiocy.

Premature ossification of the cranial suture is no longer so much regarded as a cause of idiocy, nor can we say that it alone is ever the cause of epilepsy, though its contributory effects may not be denied.

The epileptic idiot is usually small in stature and often dwarfish in appearance. He is generally undeveloped, the countenance is childish, the hair on the face, pubes, and other parts of the body is either absent, scanty, or excessive; the reproductive organs are undeveloped; the menstrual function is absent, late, or irregular; the teeth are backward in eruption and faulty in arrangement; the palate is asymmetrical. All the special senses are blunted. The deep and superficial reflexes may be either lost or increased.

There is usually incoördination or paralysis of the lower extremities. Irregular movements of the eyes, and nystagmus are common. There is stuttering, halting, or otherwise imperfect speech.

The anatomic stigmata of degeneration are found in 80 per cent. of all cases, including anomalies of the eyes, ears, mouth, nose, and especially the bones of the face, while 30 per cent. of all idiots suffer from epilepsy (Wildermuth).

These degenerate physical distinctions alone largely indicate the type of mentality we should expect to find associated with this condition. Intellectually, idiots bear some class division, being separable for educational purposes into high and low grades. It will serve our purpose, however, at this time to consider them in a single group, since we refer to epileptic idiots only, all of whom belong permanently to the irresponsible and unteachable class.

As a rule, they are wholly unable to comprehend their surroundings; possess no knowledge of their environment and manifest no interest in it, no matter how often or how radically it may change. They have no degree of self-consciousness; can form no judgment respecting the simplest matters; invariably ignore calls of the emunctories, soiling themselves habitually. They are absolutely indifferent to personal appearance.

They show no evidence of emotional life, save perhaps slight vacillation in their general feelings; rarely show any excitability or evince any feeling of pain, fear, or pleasure; they are wholly ignorant of the laws of self-preservation, save that of body sustenance, and even that is sometimes lost.

Epileptic idiocy is most frequently met with in the young, generally under the age of ten years, and rarely arises *de novo* after the eighteenth or twentieth year. Its prototype after that period is found in epileptic dementia, which may develop at any age, though it is

Plate 27.

Types of epileptic idiots; the low mental state preceding the epilepsy, the latter serving to more completely destroy what little mentality there may have been.

most common in young and middle adult life.   Like the epileptic imbecile, the epileptic idiot essentially belongs to the wholly irresponsible class.

Epileptic Dementia.—The epilepsies that remain confined in their origin during attacks and throughout the greater part of their manifestations to the motor side, seldom reduce the mental faculties to the extent of their complete destruction.   This is especially true of Jacksonian epilepsy and other forms in which the convulsion, while motor, may be general.   Many fits of the latter kind leave the psychic life in perfect integrity the moment the fit is over.

The forms of convulsions that appear to have their origin in the frontal lobes—in the "organs of the mind" —and those followed by more or less prolonged periods of automatism, or deep, prolonged coma, may induce complete epileptic dementia in favorable subjects in two or three years' time.

It has repeatedly been observed in the most pronounced cases of dementia, that the epileptic paroxysm may temporarily stimulate morbid mental commotion, with the result that the long period of sluggish apathy is broken, the patient becomes excited, violent, and dangerous, and is especially prone just then to commit murderous assaults.   This tendency stamps the epileptic dement as a dangerous factor at any moment. His frenzy appears as a flash, spends itself in a furious psychomotor storm, and disappears as quickly as it came.

Ordinarily he is quiet, obedient, innocent of harm, and plastic under control, but when the brutal (Hughlings-Jackson) expenditure of nervous force is suddenly displayed he is changed into an impulsive madman, ready to attack without motive or justification, and capable of committing the most atrocious crimes.

Epileptic dementia is rare during the senile epoch, its victims generally being between the ages of twenty

and forty years.   It is essentially incurable and tends to shorten life.   Elsewhere we have called attention to the fact that the average age at death in 220 cases was twenty-nine years.

Manic-depressive states include the acute recoverable psychoses, mania, and melancholia, under both simple and complex, and circular insanity.   It is therefore a group term used to designate the wide range of symptoms characteristic of mania and melancholia, and in which leading symptoms vary so greatly as to lead occasionally to considerable confusion.

Transitory epileptic mania is characterized by great psychomotor excitement; a purposeless activity; great destructiveness; a happy, though excessively emotional, attitude; unsystematized delusions; occasionally hallucinations; and, rarely, some dimming of consciousness, though usually the perceptive faculties are more than ordinarily acute.

The depressive forms are characterized by a diminution or loss of psychomotor activity, a lack of spontaneous bodily exertion; a paucity of ideas; an emotional attitude indicative of great dejection; a tense, drawn, apprehensive expression; prominent delusions, usually of the persecutory type, and some obscuration of consciousness.   In some cases the two states alternately appear in the same individual, and it is now generally conceded that they are phases of but one disease process.   At the same time, the constant recurrence of definite fundamental symptoms all in one attack, the uniformity of their behavior in course and outcome, the occasional intimate relation of different forms of the same disease, where one form merges slowly or rapidly into another, has led to the conclusion that all may be definitely classified under the three heads given, namely: manic, depressive, and mixed.

The two former are nearly always accentuated at the period of epileptic discharge which brings to light

Plate 28

A case of partial epileptic dementia, characterized by extreme good nature instead of marked epileptic irritability, so common after years of the disease.

Plate 29.

A case of epileptic dementia, analogous in all respects to terminal dementia following ordinary forms of mental diseases, except that marked maniacal periods are apt to precede or follow the epileptic attack.

smouldering manifestations not hitherto suspected as being present. This feature is more noticeable in the manic than in the depressive forms.

States of transitory exaltation are common, though they rarely become chronic in epilepsy; while states of depression marked by suicidal intent are distinctly uncommon.

In the writer's experience, in the care of some 1600 cases during a period of fifteen years, in 200 of which the patients were under judicial commitment as insane, he recalls but two in which the purpose of self-destruction was put into practice. In both the impulse was sudden, nothing up to the moment of the act indicating premeditation.

A woman of fifty-two years, an epileptic since her eighteenth year, who suffered from both *grand mal* and *petit mal* attacks, generally at night, and not previously depressed, jumped into a pond with suicidal intent. On being rescued, she expressed regret for the act, and four years later had shown no further inclination that way.

A young man of twenty-two years, an epileptic for two years only, whose attacks occurred at first three months apart, but later in series of from six to eight every three or four weeks, and who likewise had shown no suicidal intent or unusual depression, left his bed to which he had been confined a few days through a minor illness, crossed the hall to the toilet room, and cut his throat, death occurring some hours later.

These instances are cited more particularly to emphasize the *unexpectedness* of epileptics' acts. *The line of continuity of conduct with them in any radical way is seldom changed in a slow and orderly manner, but nearly always under a sudden impulse.* In some inexplicable way the quick, explosive violence of the fit itself seems to find parallels in action in most of the things that epileptics do out of the usual order.

We are speaking now of a characteristic applicable to the great majority. It does not include a few whose powers of inhibition seem to remain intact during the inter-paroxysmal period and even in rare instances under the duress of the lighter and less frequent forms of the disease.

Almost any number of cases might be cited in illustration of the manic-depressive states due to epilepsy. The following is fairly typical of the majority of them:

A. E. T., age twenty years on admission. Had been epileptic since fourteen. Family history good. Cause of epilepsy, masturbation. The patient was free from any stigmata of degeneration; he was of excellent disposition and good education, a high-school graduate. He always showed perfect self-control, save when under the influence of epileptic attacks, which came on at first from twelve to fourteen times a year, being alternately *grand mal* and *petit mal*. Later they averaged from eight to ten a month, while for two years before the attack of mental disturbance about to be described, they appeared three or four times only in a year.

He usually had a psychic aura, and would become greatly depressed and introspective; he was often hallucinatory for hours after the attacks, sight and hearing both being involved. All this time, during the inter-paroxysmal periods, he remained a model patient in every respect, failing to show any evidence of the effects of his disease.

Fourteen years after the development of the epilepsy, the inter-paroxysmal mental condition did not show the least sign of the disease.

In the meantime, the insane periods had been growing more intense in expression and occurred closer together. Isolated seizures left him automatic for a longer time, and his mental vagaries became more marked in proportion to the length of such periods. Series of attacks were attended with far more psycho-motor disturbances than that common to isolated attacks. While the latter produced simple automatism and mildly expressed insane ideas, the former turned him into a typical madman, with the most insane and

Plate 30.

A case of epileptic dementia, characterized by extreme loss of psycho-motor activity of all kinds—the lowest estate an epileptic can reach save that of epileptic idiocy

insistent ideas shoutingly expressed with exaggerated gestures, and forced him to assault any who came within his reach.

He had such an attack on January 16, 1903, which can best be described, in part, in his own language, which, however, fails to give any idea of the excessive degree of violence he manifested at the time.

"I had," he states, "a severe attack on the night of January 13th. I did not know it at the time, but found it out the next morning from my sore tongue, headache, and general bad feeling. I felt bad all day, but had no attack the next night; but on the night of the 15th I had several hard fits, and several more during the night of the 16th. After the last one, I was dressed (I do not recall this, but was told so) and taken to another building. I remember escaping from this building through an attic window and getting out on a steep, ice-covered, slippery roof. It was my belief that those in charge were going to kill and cook me, so I used a stick to keep them off. I also pushed the top of the ladder away from the roof to keep them from coming up to me. I heard Mr. A. tell the cook to help kill me. I saw in the person of Mr. A. a man I knew before I came to the Colony. I also heard a man say he was another man I knew at home, and I remember I did not believe him at the time. I have no recollection of seeing Mr. McC. or Dr. S. while I was on the roof." (Both of these persons talked with the patient at the time.)

Analysis of the patient's statements shows that he presented nearly every essential feature of ·manic-depressive insanity due to epilepsy. He had the delusions of persecution characteristic of depression, as shown in his fear of being killed to be eaten; he had hallucinations of hearing, as shown by the fact that he mistook ordinary conversation for commands to take part in his execution; he had hallucinations of sight, as shown by his belief that he saw a person in the crowd about him who was not there; all these being insistently engrafted upon a frenzied bodily activity, typically characteristic of the acutely insane epileptic.

His excessive psychomotor violence lasted twelve hours, gradually fading into the normal state seventy-two hours later.

It is a curious fact that he was afterward able to recall certain incidents only that transpired while his excitement was greatest. Other incidents that he appeared cognizant of at the time, as shown by the conversation he carried on about them, he was wholly unable to recall.

I have observed that the incomplete automatic state (shown to be incomplete in this instance by the patient doing things radically different from those he was habituated to perform, and his recollection of them afterward) permits the patient to recall some things, but to show no knowledge of others apparently of far greater importance.

The question of automatism in such a state as this, however, is of little moment from a medicolegal standpoint as compared with the more essential fact that the patient is so palpably and actively insane.

Very rarely we may meet with a case in which the prodromal manifestations of the fit closely simulate *circular insanity*. The similarity is not complete, however, for while the upward growth of the period of psychomotor exaltation is almost identical, the stage of decline is wholly unlike.

The period of decline in *folie circulaire* is gradual, fading conspicuously for days or weeks until the end of the cycle is reached and profound melancholia is established.

In the epileptic the cycle is suddenly cut short at the end of the upward period by the fit which abruptly dissipates all morbid psychomotor phenomena until the beginning of the next cycle, which is again broken at its acme in the manner as before.

It is safe to say (Féré, Morel, Doutrébente, Soukey) that we rarely meet with epileptics who are well

balanced mentally and intellectually.   Their character is usually mobile, and traits of depression predominate. Habitually gloomy, irritable, and jealous, they may be observed to pass suddenly to sentiments of generosity, benevolence, and enthusiasm, which vanish as quickly as the paroxysms to which they are subject.

The state of profound depression which follows the convulsive discharges presents a most favorable field for the development of feelings of sadness, inferiority, and impotence, under the influence of which suicidal impulses sometimes arise.   Their frequent hallucinations, the terrifying dreams to which they are very liable, render such persons timorous, suspicious, and distrustful.   Their tendencies often take an impulsive character, but rarely they may manifest tenacity of purpose or true energy.   The mental oscillations which are so frequent among them have given rise to the view that remittent insanity and circular insanity are manifestations of masked epilepsy.

"The lower types of epileptics exhibit a characteristic low cunning and deceit; they are treacherous in their dealings with their associates, thievish in their propensities, and when arraigned upon a charge of misconduct will meet it with the coolest audacity, and lie to the bitter end.   The epileptic shows a tendency, akin to that of the hysteric, toward maligning. Both will falsely accuse of violence those of whom they are aggrieved; will treasure up a tooth, or wilfully pull out their own hair by the handful and present it to countenance their charge, and will cunningly call to their defense certain delusional notions to which they may be prone during the period of their seizures, if they can benefit their position thereby.   This tendency should be carefully borne in mind.   All the apparent delusional statements of an epileptic are not to be received, except with caution, as their sole object may be to obtain some indulgence or request, and

especially is it so with the hypochondriacal subject"
(Bevan Lewis).

Associated with this moral decadence the epileptic
is also essentially impulsive, a feature demanding the
utmost tact in his care. His conduct when aroused is
peculiarly brutal and ferocious, and often characterized,
like his actions during periods of epileptic automatism,
by wholly disproportionate and excessive violence.

The statements of Féré, Marie, Doutrébente, Morel,
Bevan Lewis, and others, relative to the general mental
equipment, and especially the abundance of moral
decadence in the epileptic as the result of his disease,
are not overdrawn in a very large number of cases.
The picture is not a happy one. Nevertheless, too
often it is true. Many of the better epileptics, such
as usually visit the consultation room of the physician,
do not show such characteristics.

In Krafft-Ebing's *Psychopathia Sexualis* (pp. 364 to
374), numerous illustrations are found of the debased
moral practices and tendencies of many epileptics as
exhibited when apparently under the influence of the
disease. Similar cases, little less shocking, have come
under my own observation. Some such will be found
in every aggregation of epileptics of any considerable
size.

In any large aggregation of epileptics in which there
are none actively insane, scores may be found whose
conduct in every respect is exemplary.

Mental capacity in epilepsy is impaired to some
extent in the great majority of cases. Any disease
that so radically affects the brain must produce this
result. There is no method of accurately estimating
the frequency and degree of mental reduction due to
epilepsy. While certain cases naturally fall in quite
constant resultant groups in this respect, each is
finally a problem in its own right.

But there is this about every case: the most impor-

tant legal question bearing on mental reduction concerns chiefly *the duration of such reduction*. We can summarize the points of this in this manner:

1. The reduction—amounting in some cases to absolute destruction—may be measured in point of time by a few seconds only, such reduction periods being not infrequently months apart. The inter-paroxysmal condition of such patients may be perfectly normal so far as we can determine.

2. The reduction may be frequent in point of occurrence, varying from several hundred seizures in twenty-four hours to one or two in a day, or as many in a month. The inter-paroxysmal condition in such cases is generally one of permanent irresponsibility; in others in which the seizures are less frequent, there may be rational intervals.

3. The reduction may be permanent, covering both the paroxysmal and the inter-paroxysmal period, so that the individual is continuously irresponsible. The degrees of mental destruction under this head include feeble-mindedness, imbecility, idiocy, dementia, and the manic-depressive states previously described.

It is difficult to say what proportion of epileptics suffer in the lighter degree only, but it is safe to estimate the number at not to exceed from 8 per cent. to 10 per cent. These may be called sane, both within the legal and scientific meaning, *except at the brief moment of attack*.

The number coming under the second heading includes fully 50 per cent. of them all; those under the third, the remaining 40 per cent., which we may again divide into two parts, the idiots and imbeciles representing from 20 per cent. to 25 per cent., and the continuously insane (such as are recognized by law and medicine alike) from 15 per cent. to 20 per cent.

The general practitioner rarely comes in contact for any length of time with cases under the third category.

He is mostly familiar with those whose disease is comparatively recent, and of a type least destructive of mentality, so that he is called upon, as a rule, to treat only the better classes, and then he generally sees these when they are at their best, rarely being able to witness the convulsions and their effects on the mind.

Much has been written on the relationship between **genius** and **epilepsy.** I cannot feel that any disease, in which the tendency to loss of mental power is so great as in epilepsy, is capable of conferring unusual intellectual capacity. I agree with Sir Lauder Brunton when he says,* "It is quite true that some of the most remarkable men in the world's history have been epileptics, but I do not think that Julius Cæsar, Napoleon, or Mohammed were great because they were epileptics. As a rule, epilepsy tends to destroy mental power rather than to increase it, and the curious lethargy which Napoleon exhibited at the Battle of Leipzig, and which there led to his defeat and consequent ruin, is probably rather to be ascribed to his epileptic tendency than to the indigestible bun which is said to have led to the disaster. Julius Cæsar and Napoleon were great men, not because of their epilepsy, but in spite of it; and the visions of Mohammed alone would not have given him his extraordinary power over his countrymen and over the then known world, had it not been that they were backed up by extraordinary mental power and energy in the intervals between his fits."

Lombroso † devotes a chapter to The Epileptoid Nature of Genius. He says: "It is sufficient . . . . to recall to the reader the numerous men of genius of the first order who have been seized by motory epilepsy, or by that kind of irritability which is well known to supply its place. Among these we find

* "Journal of Mental Science," XLVIII, No. 201, April, 1902.
† "The Man of Genius."

Napoleon, Molière, Julius Cæsar, Petrarch, Peter the Great, Mohammed, Handel, Swift, Richelieu, Charles V., Flaubert, Dostoieffsky, and St. Paul.''

Lombroso admits that convulsions made their appearance rarely in the course of the lives of these men, and suggests that they were replaced by psychic equivalents, which, according to his views, "are more frequent and intense when motor convulsions are lacking"—a proposition I am unable to accept.

Because an epileptic fails to have frequent motor or psychomotor convulsions is no reason why he should have frequent psychic attacks.

Above all, in Lombroso's opinion, is the relationship between genius and epilepsy to be proved "through the analogy of the epileptic seizure with the moment of inspiration," and it is further demonstrated by the even more cogent proof—the confession of eminent men of genius, such as Goncourt, Buffon, Mohammed, and Dostoieffsky." The finest illustration of this to be found in the literature, perhaps, is by Dostoieffsky in "The Idiot," Vol. I, p. 296.

"I remember," says the author (evidently referring to himself), "among other things a phenomena which used to precede his epileptic attacks when they came in the waking state. In the midst of the dejection, the mental marasmus, the anxiety, which the madman experienced, there were moments in which all of a sudden the brain became inflamed, and all his vital forces suddenly rose to a prodigious degree of intensity. The sensation of life, of conscious existence, was multiplied tenfold in these swiftly passing moments. A strange light illumined his heart and mind. All agitation was calmed, all doubt and perplexity resolved itself into a superior harmony, a serene and tranquil gaiety, which yet was completely rational. But these radiant moments were only a prelude to the last

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instant—that immediately succeeding the attack. That instant, in truth, was ineffable."

Later on when the attack was over, the author's reflections ran thus: "Those fleeting moments in which our highest consciousness of ourselves—and therefore our highest life—is manifested, are due to disease, to the suspension of normal conditions; and, if so, it is not a higher life, but, on the contrary, one of lower order."

Strangely enough, he then goes on to say: "What matter, after all, though it be a disease—an abnormal tension—if the result (as I will recover health, remember, and analyze it) includes the highest degree of harmony and beauty."

Lines from the correspondence of Flaubert contain the following expressions significant of the acute epileptic state. "I who have heard through closed doors people talking in low tones thirty paces away (hallucinations of sound), across whose abdomen one may see the viscera throbbing, and who have sometimes felt in the space of a minute a million thoughts, images, and combinations of all kinds throwing themselves into my brain at once, as it were a lighted squib of fireworks."

The confessions of Dostoieffsky and Flaubert are similar to feelings described to me by epileptics of superior intellectual endowment. Two in particular detailed the sensations of intellectual aura they experienced, which would readily pass for these confessions. They had such aura rarely, and declared it to be "the most overwhelming ecstatic state it is possible for the human mind to conceive of." Both were teachers of noted ability and both developed epilepsy through excessive alcoholic indulgence—not drinking enough at one time to produce drunkenness, but drinking systematically for years to fortify a nervous system exhausted through overwork.

No less interesting than the foregoing are Sweden-
borg's insane manifestations due to epilepsy.*

Always eccentric to a marked degree, it was not
until his fifty-fifth year that his particular malady
became conspicuous.

Authenticated instances in detail are cited by
Maudsley, in which Swedenborg passed through all the
phases of the epileptic convulsion, even to frothing
at the mouth.   A record in his diary is this: "There
happened to me something very curious.   I came into
violent shudderings, as when Christ showed me His
Divine Mercy.   The one fit followed the other ten or
fifteen times."   Apparently this meant serial epilepsy.

"In 1774, when he was fifty-five years of age," says
Maudsley, "Swedenborg suddenly abandoned his
former pursuits and interests.   He claimed to have
been admitted into the spiritual world, and that he
possessed the power of talking with angels.   Coinci-
dently with this great change and new missions, he
was entering into what an unprejudiced person must
affirm to be the product of madness; this condition
finally developing in unmistakable form."

"Is it not reasonable," asks Maudsley, "to infer
that those new and strange pictures were the outcome
of his madness?   His disciples say not, but those
familiar with the product of diseased epileptic fancies
see nothing but epileptic phenomena in them."

Because of the frequency and extent to which epi-
lepsy impairs the emotional balance, for the same reason
apparently it stimulates the creation of delusions,
hallucinations, and illusions that partake of a religious
order, or are wholly religious in their expressions.   So
long as such phenomena remain free from interpreta-

* We only touch upon the vagaries of this unique character in this con-
nection, and suggest to those who wish to pursue the subject further, the
reading of Maudsley's "Body and Mind," and William White's "Emanuel
Swedenborg: His Life and Writings."

tion by the patient as constituting divine commands, the individual is less likely to do violence than when he feels impelled to carry out such commands. It is never wise to assume that epileptics of morbid religious tendencies are safe to be at large. Divine commands appear to them as sudden inspirations and generally lead to acts of great violence. Without exception such persons require the restraint of hospitals for the insane.

L. B., a young adult epileptic, proclaimed these sentiments for months: "God has told me that in my next life I would be born of C. H. S. and marry my last sweetheart, and be a millionaire, and that I would be a strong, hopeful, good, powerful Christian millionaire. I feel God's voice in my left ear at night. I feel the Lord in my chest. I see stars in my eyes during the day, which are the Holy Spirit. I have seen Christ crucified. I am cured of my fits; I have worked them off by asking the good Lord to forgive my sins. God told me I would never have any more fits. God told me to bite off a patient's ear. If God told me to do wrong, it feels as if I would do it. God told me the world was coming to an end very soon. The Holy Spirit has been working on me twenty-two weeks."*

It has often been observed that the nature of the delusion bears some relationship to the character of the mind from which it springs. The form of the insane person's vocation is therefore likely to be reflected in the genesis of his delusions. It seems as reasonable to picture the expression of morbid, exalted, and ecstatic states encompassing a wealth of perverted intellectuality as coming from minds of great natural endowment, such as Mohammed's, Swedenborg's, and others,

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* Shortly before his commitment to a hospital for the insane, he endeavored to obey a "divine command" by almost biting off a fellow-patient's ear.

as to picture more commonplace delusions as springing from minds of inferior capacity. Had Swedenborg or Mohammed been actively epileptic in their youth, probably neither would have stamped his individuality upon history as he did.

We are told that the picture given the world by Taine is the completest view of Napoleon ever given by any historian. "To any one acquainted (we quote again from Maudsley) with the psychologic constitution of the epileptic it becomes clear that Taine has given us the subtlest and most precise pathologic diagnosis of a case of psychic epilepsy with its giant megalomaniacal illusions, its impulses, and complete absence of moral sense."

"I see no reason," says Professor William M. Sloan, author of a most admirable and complete "Life of Napoleon," "to question the fact that Napoleon I was an epileptic. There were, I think, two instances when he had short seizures which did not amount to fits, but were regarded by contemporaries as symptoms of epilepsy."

It seems evident that such attacks were either *petit mal* or psychic, similar to those experienced by Julius Cæsar. In Appian's "Roman History" we find this reference to Cæsar: "At length, whether he lost all hope, or else for the better preservation of his health, never more afflicted with the falling sickness and sudden convulsions than when he lay idle, he resolved upon a far distant expedition against the Gatæ and the Parthians."

In Seutonius's "Lives of the Cæsars" we also find the following concerning Julius Cæsar. "He is said to have been tall, of a fair complexion, round-limbed, rather full-faced, with black eyes, and lively; very healthful, except toward the end of his life he would suddenly fall into fainting fits, and be frightened in

his sleep.   He was twice seized with the falling sick-
ness in time of battle.''

We might continue to pursue the study of men of
genius written in history as epileptics in a far more
elaborate way, without learning anything more convinc-
ing than the facts apparent in the cases cited, namely,
that none of them had *essential* epilepsy in early life, so
far as history goes to show; that they all developed it
late in life in comparatively mild forms—in forms not
incompatible with the highest intellectual endowment;
*which latter, however, must be developed in full before
the establishment of the disease*, and which the true dis-
ease tends in every case to impair or destroy.

CHAPTER XVII.

THE MEDICOLEGAL ASPECTS OF EPILEPSY.

Pre-paroxysmal Forms of Mental Disturbance. Religious Emotionalism. Transitory Epileptic Irritability. Paroxysmal Epileptic Insanity. Post-epileptic Automatism. Characteristic Conditions under which Epileptics Commit Homicides. Cases in Illustration. Epilepsy and Life Insurance.

The medicolegal problems in epilepsy, like those of insanity, relate to the mental condition at the time of the commission of a crime or act, or voicing of an expression, for which the accused person cannot rightfully be held responsible, and to charges brought by epileptics against others for injuries sustained during an attack. It may also be a question at law as to an injury resulting from actionable negligence in a case of epilepsy.

While the insane manifestations in epileptics often partake of the same general characteristics, such as explosiveness, great violence, and brutality, there is no rule by which all cases can be studied alike.

We have previously noted that epileptic seizures of certain types may occur for many years without appreciably impairing the mental faculties, while others destroy the mind in much less time.

We can best study mental reductions incident to epilepsy and its correlated question of responsibility by considering them in their relationship to the seizure periods, *i. e.*, whether they precede, accompany, are substituted for, or follow the attacks, or whether the insane state exists continuously irrespective of the epileptic "discharges."

Pre-paroxysmal Forms of Mental Disturbance.—The psychic aura that sometimes appears days before the

seizure may be often regarded as a species of insanity. Persons in this state, as a rule, know the nature and quality of the wrongful acts they commit, but they suffer so complete a paralysis of the powers of inhibition that they are incompetent to restrain themselves. Others have no knowledge of what is going on about them when in the deeper reduction of a psychic aura just before the fit.

An intelligent woman of forty-three years, who was under my care for some years, had an unusually pleasant disposition, except about the times of her seizure periods. For days before these, she would be mildly excited, loquacious, alternately crying and laughing, complaining one moment of fancied evil remarks made about her, and the next vehemently asserting that the fault all lay in herself. She was nervous, restless, irritable, and sleepless; she complained of strange cephalic sensations; her face was flushed, her eyes unnaturally bright, and her gait unsteady to the point of stumbling. She described her feelings while in this state as follows:

"This is now the 22d of February, 1902. I had four very severe attacks on the 31st of last January. I felt the attacks coming for a long time before they came. I was exceedingly nervous and irritable; I scolded about everything and was unable to hold things in a proper manner. When I picked up my brush and comb, I would drop them, and when I walked I stepped crooked—one foot would keep making mistakes and I didn't know which foot it was. They said it was the left. I could not sleep at all. I lay thinking of things that had happened and that were not pleasant; I tried to put them out of my mind by thinking of what I had read. I have a strong will and feel that I can control my thoughts at most times; *but for days before the attacks come on, I am unable to do so*. They go off about everything, and I try to make them come back, but they won't. They get worse every day and night, and finally they get all jumbled up together, and I don't know anything. Then the attacks come on, and after that I am all right—everything clears up and I see things as they

really are. I lose all my nervousness and can act rationally."

During the greater part of this morbid state, the patient was not only conscious of what she was doing, but knew full well the import of every act. Several times while in this state she made groundless complaints against the nurse, which she retracted either immediately or as soon as the fit had passed, declaring they were unjust. The main point is that she was powerless to inhibit such conduct. The line between this state and insanity is delicate in the extreme. Scientifically it is insanity; legally, it might not be so regarded.

The principle of freedom from responsibility through defective inhibition was sustained by Judge H. M. Somerville, of the Supreme Court of Alabama, in 1887. He rendered a decision that repudiated the test of insanity laid down in the celebrated McNaughton case in England. The test in this case was the ability to distinguish between the right and wrong of the act in question. Judge Somerville held that the true test of responsibility in cases of insanity is the power to refrain from doing the act; in other words, the question to be decided was, whether the alleged evil-doer acted under duress of a disease which had impaired or destroyed his powers of inhibition.

Paradoxical religious emotionalism is apt to be manifested by epileptics just before the seizure periods and should always be regarded as a danger signal. Clouston * mentions a lad in whom it was a sure prelude to a fit or a series of fits. Before these periods, the patient read his Bible continually, and when spoken to answered fiercely, "Don't trouble me; I am a good man; I'm a servant of God." The day after he would walk up and down and strike any one who came near him. If any one spoke to him, he replied

* "Mental Diseases," 1884, p. 288.

maniacally, "You are a d——d liar.   Don't insult me."
In a few hours he would have one or more fits, remain
stupid for awhile, then be as well as ever.

A man of forty-three years, under my care, whose
epilepsy had followed scarlatinal nephritis at the age
of seven years, and who was subject to long remissions
in his disease, had serial attacks from three to four
weeks apart.   The first indication noted of his ap-
proaching fits was his fault-finding at the table.   He
suddenly objected to his neighbor, calling him a vile
name.   At the next meal he refused to sit beside him
and at the next meal he failed to appear at all.   He
was found in his room shortly after, moody, sullen, and
irritable, reading his Bible.   He kept this up all night
and the better part of the following day, when he
suddenly lay his Bible aside and began to loudly
revile everyone within hearing, in the most profane
and violent language.   On his finally attempting to
assault his nurse and physician, he was placed in
restraint.   A few hours later, he had three severe
attacks in rapid succession, six hours after which he
was composed and agreeable to all about him.   His
malady followed this course for many years.

Comparatively few epileptics have such a distinctive
psychic aura.   In 1325 cases under my observation,
33 men and 19 women manifested it in some form—
most of them to a less extent.   Its relative infrequency
makes overt acts performed under its influence rare.

Such epileptics should not be regarded as mentally
sound at any time.   They often experience subcon-
scious automatic states in which they display no
violence, in which they are wholly without conscious
volition.

Transitory Epileptic Irritability.—I have previously
spoken of the effects of epilepsy on the disposition.
These are especially noticeable before the fit.   The
patient becomes querulous, exacting, fault-finding;

nothing suits him; he fancies his friends untrue, and charges his family with deserting him.   He impugns the motive of all about him; is incapable of continuous application; unable to sleep well, and has terrifying dreams.   Morbid ideas finally assume complete mastery over him.   As a rule, these ill-humor periods end in a convulsion.   They may come and go independently of typical seizures.

There should always be a doubt as to the responsibility of epileptics who commit wrongful acts while in this state.   In some it is impossible to say that such a state is not the result of a preceding unrecognized convulsion.

Epileptics not infrequently assault those about them when in this humor.   In any large epileptic community, such events are of daily occurrence.

Innumerable instances have come under my observation in which epileptics have charged their attendants with brutal conduct, and in which investigation showed the charges to be either malicious attempts to injure others or the result of the perverted sensations so common in epilepsy.   I have found the latter more often the case than the former.   This is readily understood.

When a fit is about to appear, the patient is seized with the most terrifying apprehensions.   He is in an agony of fear.   All his senses are stirred to a profound degree and he is imperfectly conscious of what is happening about him.   An attendant grasps him to assist him.   When the fit is over, feeling bruised and sore as the inevitable result of the attack, he accuses the attendant with having assaulted him.   I have also known numerous instances in which the patient declared that harsh language had been used toward him, his conception being due to hallucinations of hearing—not a word having been spoken by those about him.   Others, on suddenly experiencing a dis-

tressing ringing in the ears just before they become unconscious, and being injured about the head by falling during the fit, afterward declare that some one struck them on the head.

The most common complaint is based on the feeling of suffocation the patient experiences just as he is passing into oblivion, and which he very frequently says is due to some one kneeling on his chest purposely to injure him. While all epileptics do not have these periods of ill-humor and hallucinations, the majority do. To prevent injustice to innocent persons, epileptic testimony needs to be received with great caution.

Paroxysmal Epileptic Insanity.—Unquestionably the gravest criminal result of epilepsy is the commission of assaults and inhuman crimes during the fit itself or during the automatic state that immediately follows it.

From the earliest times its mental accompaniments have increased the mystery and terror of epilepsy. When added to the contortions and unconsciousness of that disease during a fit, there are afterward developed strange hallucinations, terrible acts of impulsive violence, and striking religious delusions, we cannot wonder that a supernatural cause was almost universally believed in of old.

"I have seen epileptic insanity take the form of a more acute maniacal condition than almost any other insanity, with the exception of mania from alcohol. There is no other form of insanity outside of asylums so frequently the cause of murders" (Clouston).

"The excitement in epileptic mania is most acute. No maniacs show such blind, incalculating violence and frequent fury as the epileptic. He is one of the most dangerous subjects we have to deal with in our asylums. The aspect of the patient fully accords with the impulsive conduct. He is usually pale and ghastly, the eyes staring vacantly, and the face expressionless or

betraying wild and passionate emotion" (Bevan Lewis).

"The nervous excitability may increase as the time for the attack approaches, and before its occurrence a maniacal outbreak of great violence may take place, during which the most brutal and wantonly cruel acts may be committed. Such acts may be against relatives or best friends, but sometimes persons with whom the epileptic has not been on the best of terms suffer, thus giving the deeds of violence a superficial appearance of having been premeditated" (Peterson and Haines).

The following cases from the literature and from my own experience show in some detail the nature of such acts and the circumstances under which they are usually committed:

Case I.—P. P., a young man of twenty-four, a native of Greece, was indicted and tried for murder in the first degree for shooting to death his partner in business. Competent testimony was adduced to show that the defendant and the man he killed had been the best of friends for years; that there was absolutely no motive for the crime; and that the defendant not only came from an epileptic family, but was himself a victim of the disease.

On its face, the murder bore every evidence of a cold-blooded crime. In the presence of witnesses the two men quarreled while trying to adjust a small account. Angry words passed between them, and ugly insinuations, derogatory to the fiancée of the defendant, were made, when he suddenly drew a revolver and fired five shots pointblank at his partner as fast as a self-acting revolver would work, three of them taking effect and causing death.

Witnesses testified that the defendant then threw his revolver on the floor and made no effort to escape. It was also testified that he was heard shortly afterward to say, "It's too bad; it's too bad!" The shooting occurred about a quarter past twelve in the afternoon, and the defendant was at once lodged in jail. Other witnesses testified that after the defendant was

in jail and when asked what he was doing there, replied, "I shot George."

In his own testimony on the stand the defendant declared that his mind was a blank from the time of the quarrel in the store until he came to his senses in the jail about two hours and a quarter later, and that he knew absolutely nothing of what transpired in that time.

The murder was committed in April, 1901, and the trial was held in February, 1902. In the meantime, two certificates—one from a physician, the other from a priest—were secured from Greece, to the effect that the defendant's father was an epileptic; that he often had "nervous sickness" and would "fall down almost daily," and that he had been so afflicted for many years.

I examined the man and gave testimony in the case, and repeated examinations made while he was in jail revealed, among other things, the following:

He was born in Greece and came to America nine or ten years previously. He spoke English quite perfectly and understood readily; he answered questions in a simple and straightforward way. Age, twenty-four; height, 5 feet two inches and a half; weight, 134 pounds. Present occupation, flower merchant. His mother died in childbirth; his father is living and subject to epilepsy; his grandparents lived to old age, the father's mother dying of epilepsy. He has one sister living and well. There was no history of insanity or intemperance in any of his ancestors obtainable, although it was stated that his father became mentally disturbed at times as the result of his epilepsy.

Physiognomy: The complexion was clear and smooth; nutrition was good; the expression was calm and benign; speech was clear and distinct, except that the English was not perfect. There were no anatomic stigmata about the face, cranium, teeth, palate, ears, or limbs. There was some anomaly of skin in the way of dense hairy growth over the shoulders, chest, and arms. There were no physiologic stigmata in the way of tremors, tics, or nystagmus; nor was there any evidence of mental stigmata of any kind. He did not appear to be erratic in any way; was not emo-

tional or egotistic.   All superficial reflexes, including the plantar, cremasteric, abdominal, epigastric, and scapular, responded naturally.   Of the deep reflexes, that of the left patella was somewhat exaggerated, those of the wrist and elbow being normal.   The left pupil responded more slowly to light than the right. There was no ankle-clonus and no impairment of tactile sensation in any part of the body.   Vision and taste were normal; hearing was defective, especially on the right side.   Measurements of the limbs showed nothing anomalous.   Power of grip was coequal, but low in both hands.

I was constantly impressed with the man's evident honesty and sincerity, and his expressions of regret. for the crime appeared to be genuine.

He stated that the first epileptic seizure he had, that he remembered, occurred when he was eight years old, at which time his father told him of a former attack that had occurred when he was six years old. On one occasion he fell from a single story window while in a fit, cutting his head in several places; there are three small, star-shaped scars on the forehead a little to the left of the center, and a fourth one in the edge of the hair above the left temple.   He described an attack that occurred in Middletown, Connecticut, in 1895, when he fell downstairs, and others he had in jail after the murder, the first occurring three weeks after that event.   The jailer testified that he saw the defendant three different times in attacks of some sort, and, from what I could learn of them from the jailer, it was my belief that the attacks were epileptic.

The defendant testified on the stand that the last thing he remembered in the store—where the shooting was done—was a feeling as though "a wave of cold air was creeping from his feet upward," then his eyes "got dull and flew about"; he saw "yellow, red, and green," after which he remembered nothing.   He also declared he experienced the same sensations on the occasion of the attack in Middletown in 1895.

The question arose as to the mental condition the patient was in when he fired the shots.   I stated it as my belief that if the patient experienced the initial symptoms of an epileptic convulsion, such as he

described as being present at the time, he was in an automatic or subconscious state when he committed the deed and could not, therefore, be held responsible.

The question then arose as to whether a person in a *complete* state of epileptic automatism could receive a mental impression that he could retain and carry into the conscious state beyond, and I gave it as my opinion that such a thing would not be possible.

Then came the question, "How could the defendant realize he had done something wrong and seem to regret it (as it was claimed this man did, when he said shortly after the shooting, 'It's too bad; it's too bad'), if he was in an automatic state at the time?" My contention was that even though the defendant might have dimly realized at the time that he had done wrong, it was no evidence of the possession of his mental faculties, for while in such a state a person may be spoken to and will correctly respond, yet retain no knowledge of it when the subconscious state is gone.

After prolonged deliberation, the jury returned a verdict of manslaughter in the second degree.

If this man was an epileptic and was under the influence of a seizure at the time he committed the deed, he should not have been convicted. But in such cases we encounter a somewhat anomalous situation, for while the law recognizes the absence of responsibility in insanity, it fails to do so in epilepsy, and if this man had been found insane, he would have been committed to a proper institution, but being an epileptic, he must either be found guilty and made to suffer for his act, or be acquitted and allowed to go free. Had his defense been insanity, the verdict, in all probability, would have been "guilty, but insane."

Case II.—C. F. C. In the "Journal of Mental Science" for July, 1901, Percy Smith, physician for mental disorders at the Charing Cross Hospital, reports in detail the case of a man who murdered his wife and child during the night, who was found the next day at noon sitting on a chair in the middle of the

room in which the bodies lay, with his night clothing still on, his eyes closed, his head bent forward, and his hands loosely in front of him. An empty vial which had contained chloroform and oil of cloves was found on the washstand, and there was a smell of the latter drug. There were blood-marks on the prisoner's clothing and on his arm.

He seemed unconscious, made no reply when spoken to, shouted at, or shaken, or any resistance when he was laid on the floor that he might be dressed to be taken to the police station.

On arrival at the police station at 3.15 P. M., further attempts were made to arouse him, and with the idea that he had taken some poison, a stomach-pump was passed, but the fluid that was drawn off merely contained clear gastric juice and did not smell of either chloroform or oil of cloves. At the same time he is reported to have said: "I have been roughly treated; which of my children is dead?" Two hours later he was examined again and appeared to be thoroughly conscious of the position he was in, and to be sane, but said he was entirely unconscious of anything that had happened between the time he last applied the chloroform and oil of cloves after going to bed, to relieve a severe headache, and the time when he became conscious at the police station after the stomach-pump had been passed.

He burst into tears and used many expressions of endearment in regard to his wife and child, adding, "We never quarreled in our lives. How could I have done it? I suffer very much from my head, especially at night."

He was fifty years of age, and had entered the army in 1870, serving for many years in Egyptian campaigns, and he had been known during that time to suffer from "*petit mal de soleil*" and "*fainting spells.*" As a soldier his conduct had been exemplary, and he had risen to the highest rank obtainable by a non-commissioned officer.

It was also reported that he had a sunstroke shortly after entering the army, which rendered him unconscious from noon one day until the following morning, and that he became subject to "fits" soon after that.

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The physicians called to examine him endeavored to establish his irresponsibility on account of epilepsy.

As previously stated, in his charge to the jury the judge did not refer to the rules in the McNaughton case, or mention the question of "right and wrong," "a knowledge of the nature and quality of the acts committed," but stated that practically the only question the jury had to consider was whether the deed had been committed under circumstances which would absolve the prisoner from the full consequences of the crime; adding that when the jury was considering the history of the prisoner they could not shut their eyes to the history of the other members of the family.

The jury returned a verdict of "guilty, but insane," and the prisoner was ordered to be detained during the pleasure of the Government.

In marked contrast to the enlightened jurisprudence principles manifested in this case, are those in the following:

Case III.—(Reported by Wise, "American Journal of Insanity," Vol. xlv, p. 360.)   R. B., a native of England; emigrated to the United States at the age of nineteen years.   He was indicted for murder in the first degree on March 22, 1888, being at the time twenty-seven years old.   His victim was a married woman, the wife of his friend.   His crime was characterized as "one of the most atrocious and fiendish murders ever committed in Tompkins County."

R. B. was known to have affection for his friend's wife.   Though frugal, he bought her expensive presents.   He left his house one evening to visit M. and his wife.   M. found him later at the back door of his (M's.) house and invited him in.   They engaged in conversation for an hour, M. asking him to stay over night.   His wife had gone to bed in an adjoining room.   M. was standing paring an apple to eat, and started to pass B., when the latter made a sudden assault upon him.   M. afterward testified: "He struck me on the back of the head three or four times and knocked me down with something I did not see.   He knocked me senseless on the floor.   It seems to me I got up, turned to him, and said, 'Did you strike me?'

and he said 'No,' just as calm as could be, and I did not know where the blow came from.   Then he struck me three or four times and I fell in an opposite direction.   B. then went to my wife's room and began pounding her.   She hollered murder and screamed quite loud four or five times.   I heard him continue pounding her."

M's. evidence made it appear that B. returned to his wife's room, repeated the assault upon her, then returned to M., who had crawled under a high-legged bureau.   M. spoke to him but received no reply.   B. then threw the hearth rug and some cushions over M's. legs, poured kerosene oil from the lamp over them and set them on fire.   M. kicked them off.   After watching the flames until they got good headway, B. left the house and was next seen on the highway less than a mile from the burning house.   M. crawled out of the burning building to the wood pile in the orchard, where he was found by neighbors who had been attracted by the fire.

A man in a buggy discovered B. and asked him to get in and ride to the village to a dance with him. B. made no effort to escape, but accepted the invitation.   He was immediately arrested without resistance, taken before M. and identified.   Threats to lynch him did not disturb him in the least.   When asked why he struck M. and his wife, he said, "I do not remember doing it," sticking to this declaration throughout.   He finally said, "I must have done it, as everybody tells me I did."

He stated that the last incident of the evening he remembered was eating apples with M.   The remains of the murdered woman were found in the burning house.

The line of defense was based chiefly on an inherited epileptic diathesis, the prisoner having been epileptic until the age of nine years; on symptoms of nocturnal epilepsy which had appeared during the preceding winter; and on the absence of motive.

The hypothetic question of the defendant's counsel is presented as showing the substance of the direct evidence bearing upon the prisoner's responsibility.

Question: "The defendant, R. B., is twenty-seven

years of age, and unmarried. He was born at Billing-boro, England, and there resided until nineteen years of age. Since that time he has resided in the vicinity of Trumansburgh, N. Y.

"Defendant's great-grandfather had hemiplegia, or paralysis, and was to a certain extent maniacal previous to his death. His grandfather was affected with epilepsy and during one of these attacks inflicted great injury upon a friend who was trying to restrain him. His father's brother was an epileptic, and died by falling into a ditch during an epileptic fit. His aunt was an epileptic, became insane, and was confined in a lunatic asylum at Lincolnshire, England. Another aunt was a confirmed epileptic. His cousin developed epilepsy at the age of twenty, and had had epileptic fits many times since; and when he had them, it required several men to hold him until the attacks were over. Another cousin's two children had been subject to epilepsy. His grandfather's cousin had been subject to epileptic fits, and committed suicide by hanging. The above-named persons were very violent during the attack of epilepsy. His oldest sister died at the age of two years in an epileptic fit. His sister next younger than the defendant died at the age of ten months, in an epileptic fit. His brother, aged twenty-three, had epileptic fits occasionally, up to the time of his leaving England three years ago. His sisters, aged respectively twenty and eighteen, had suffered severely from fits until about eight years of age. His brother, aged thirteen, also had had fits until he was about eight years old. His brother, aged twelve, had been subject to fits all his life, and these fits were very violent. His brother, aged nine years, had had epileptic fits until he was eight years old. His cousin had been subject to epileptic fits. The defendant, R. B., had had fits, which had been accompanied by delirium and violence during the attack and for a short time after, almost weekly and sometimes several in a week, until he was nine years old. That all the above-named family were at times highly nervous and excitable. His grandfather and aunt were especially excitable, passionate, and impatient of control or contradiction. That a simple indisposition in the above-named

family—feverish, stomachic, catarrhal or otherwise—caused extreme nervousness, violence, and delirium. That these convulsive attacks rendered said B. temporarily maniacal, followed at times by great mental prostration; that he was always very violent during these epileptic attacks and had to be restrained by force to prevent his doing an injury to himself and others; the most violent part of the attack lasting about fifteen minutes and the entire attack about an hour or an hour and a half. While in England the said B. was treated a great many times for epilepsy and convulsive seizures by Thomas Blasson, a physician. He had over four hundred of these attacks before he was nine years old. The said B. while living in England displayed a good-natured, pleasant disposition, was very kind-hearted and a good, attentive, affectionate son and brother. He was temperate, steady, a regular attendant at church, and an industrious workman. During the eight years B. resided in this country, he had worked industriously most of the time out of doors; had been regular and temperate in his habits, and was much respected by his acquaintances; had never committed or was accused of any crime, previous to the present one. During the winter, and a year or two before, he complained a good deal of pain in his head, and was somewhat abstracted and moody and did not associate with young people to any extent. He could not sit in a warm room for any length of time, and used to sit in a cold room or go to a chamber room by himself. The sheets of the bed during the winter were frequently soiled with blood and water, and the bed was frequently stained, as though wet with urine and semen. During the winter of 1887–1888, the defendant had been afflicted with incontinence of urine during the night, although to no great extent.

"That he had been in jail since the 17th day of March, and that while in jail he was observed to have an epileptic fit, or *petit mal*, or light epileptic seizure, and had displayed some fury.

"Fully considering the above facts, the medical history of his family and of the defendant; the character and details of the crime; the acts of the prisoner

subsequent thereto: What would be your opinion as an expert as to the condition of the accused at the time of the commission of the crime?"

Eleven reputable physicians testified in substance that the prisoner, in their opinion, was in an unconscious, epileptic, or automatic state and was, therefore, not responsible.

One of the two physicians for the prosecution said the prisoner "might or might not be an epileptic"; the other declared the question did not change his views—he thought the prisoner sane.

In his charge to the jury the judge said: "You will therefore see that there may be a very broad difference between what medical men define as insanity and legal responsibility. No matter how insane a man may be, no matter how much under the influence of an epileptic attack, or epileptic furor, no matter by what force impelled resistible or irresistible, if this defendant at the time he did the act knew the nature and quality of the act, and knew that it was wrong, then, gentlemen of the jury, he is, in the eye of the law, legally responsible for the act that he has done, and if that act constitutes a crime, he must suffer the punishment which the law prescribes. . . . That it is not necessary for the people to show to you that there was an adequate motive for this act. It is not necessary for the people to show you what his motive was, but they claim that the reason and the method and the plan and design, apparent in the act which he did, in themselves indicate sanity, and show that there was motive for the act itself."

Notwithstanding the judge's clear and concise instructions to the jury, they returned a verdict of "guilty of arson in the first degree." They were reinstructed and rendered a second verdict of "guilty of murder in the first degree."

In polling the jury, one member in answer to a question, said, "Yes, I think her death was caused by burning." This is referred to to show the confusion in the minds of the jury caused by the intricate question they were called on to consider.

The prisoner was sentenced to be hanged on December 18, 1888. His sentence was later commuted to life

imprisonment, and he is now (October 1, 1903) in prison at Auburn, N. Y.*

Case IV.—Marie Barbella, a young Italian woman, murdered her lover apparently in cold blood by cutting his throat with a razor in New York in April, 1895. She was tried, convicted, and sentenced to death in the electric chair, but so much public interest finally crystallized about her case that she was granted a new trial, which resulted in her acquittal and discharge from custody. The plea of the defense was insanity from epilepsy.

I give the full substance of the hypothetic question asked of the expert medical witnesses for the defense, for it shows not only the condition the defendant was apparently in prior to and at the time of the crime, but indicates the pains taken to establish her epileptic ancestry. The full elucidation of the family history is always a matter of vital importance in medicolegal cases of this kind.

Question: "Assume that a woman, X, was born about twenty-four years ago in Italy; that she was the daughter of poor and illiterate parents; that her paternal grandfather was a drunkard of a low order of intelligence; that her maternal grandfather died about forty years ago, and that he was observed on a number of occasions to be seized by fits in the street, when he would rush against persons and attack them. That these attacks were observed three or four times at least, and that nobody provoked such acts, so far as the observer could ascertain. That when he had his attacks he assumed a threatening position, but when he had no attacks he was a very considerate man of severe and earnest appearance. Assume that the maternal grandmother of this woman X had an attack of some kind about a year before her death, which

* Dr. Thomas Blasson, mentioned in the hypothetic case, wrote to the "American Journal of Insanity" about R. B., March 24, 1889, in part as follows: "I know the whole of his family and its pedigree, and I can positively state that a more complete *vis consanguinitatis* I never saw. I brought this poor wretch into the world (as the saying is) and I have been the medical attendant of his grandfather, grandmother, father, mother, aunts and uncles, cousins and second cousins, for thirty-three years, and I never knew a family history so charged 'up to the hilt' with direct hereditary cerebral diseases."

occurred during the night; that she was found the next morning with her mouth distorted and unable to speak and also unable to walk. That this condition continued for some two or three months, when she gradually recovered. That about a year after the first attack she had a similar one, after which she lingered for about fifteen days in a condition like that after the first attack, when she died. That during her life she was much addicted to the use of intoxicating liquors, being often in a state of drunkenness.

"Assume that the maternal grandfather had one brother who was a hunchback, and another who was an habitual drunkard, and who used to smash furniture and everything that he could lay a hand on, and also threatened the life of his wife.

"That the mother of X had one brother, who died in convulsions at the age of four years. That the father of X was one of six or seven children; that he had one brother, now living, who was an habitual drunkard; that this brother had a habit of stripping off his clothes and exposing his nude person in the streets, sometimes when drunk and at other times when sober; that he had, on a number of occasions, piled up the furniture and other household articles in his house and set fire to them.

"That of the sisters of the father of X, one, for a very slight reason, used to strike herself on the face with her fist, pull her hair, throw herself on the ground, and turn over so as to strike her head against the walls. That the paternal grandfather of X was a man accustomed to talk unintelligently and irrationally, and, when spoken to appeared to have no understanding of what had been said. When told to do a thing, would do exactly the opposite.

"Assume that the mother of X was forty-seven years old; that she was married at the age of fifteen; that she suffered from headache and 'a turning of the head,' so that she became unable to talk; that she often had to take hold of something to keep from falling. That there were moments when her eyes were in utter darkness with streaks of red and white that flash before them; that she sometimes had cramps in the muscle of the right leg, and then a general painful

contraction of the right side, up to the neck, which lasted a quarter of an hour and more, and that up to within a few years ago she was accustomed to have attacks in which she would fall to the ground, have convulsions, and while muttering would shift her tongue from one side of the mouth to the other; that at such times her eyes would be wide open and she would stretch her arms and legs out stiff; that her face would be pale when thus attacked and she would be stupid when recovering from the attack; that when such an attack was approaching, she would often cry and then fall to the ground.

"That these seizures came at irregular intervals, sometimes being several months apart, and again at much shorter intervals.

"That the mother of X has had thirteen children and two miscarriages, seven of the children being dead, and all dying in convulsions before the age of two years.

"That the father of X was about fifty-eight years of age; that during his early manhood up to about his thirtieth year he had been an habitual drunkard; that he was illiterate and of a low order of intelligence. That the eldest brother of X was twenty-eight years of age; that up to his seventh year he had been liable to convulsions; that one day when about twelve years of age he was attacked by convulsions and fell, striking himself so that his face bled; that he was put to bed and when asked what the matter was, said he 'did not know anything'; that when he lay on the floor he was of the color of death, his mouth was distorted and frothy, and he was in a trembling condition; that after he was put on the bed, sleep intervened and lasted two or three hours. When he awoke and was asked what the trouble was, replied, ' I do not know anything, I think I had a turning of the head which caused my accident.' That after this attack his clothes were found wet from micturition. That he had always been a light sleeper, complaining of pains in his head and dizziness.

"That the brother of X next to the one just now described, was now twenty-one years of age; that he could not walk until about the age of three; that as a

child he had convulsions, and when about seven or eight years of age he suddenly fell on the ground and hurt his head, so that he had to be picked up and cared for; that after he had been put upon a bed he slept several hours, and when asked what was the trouble, was unable to respond intelligently; that he had had three or four such attacks; that he went to school for about three years and left because it was too difficult for him to learn; that he was subject to 'turnings of the head'; that he was a light sleeper, often not falling asleep until morning; that he was troubled with terrifying dreams—he constantly heard something like bells ringing in his ears, and his memory was very weak.

"That the youngest brother of X was fourteen years old; that when an infant he was afflicted with convulsions; that once when about eight years of age while at play, stars suddenly came into his eyes, after which he could not see, and fell.

"That X had a sister, now about twenty-three years of age, who, from childhood up to the age of ten, had suffered with convulsions; that when about fourteen years of age and engaged in washing, she had 'a turning,' 'a dizziness,' and fell; that after the feeling of dizziness she could not see, and when she got up knew nothing of what had happened; that once she was holding her little brother in her arms, when a dizziness overcame her, her arms loosened, she could not see, and let the child she was holding fall; that it was half an hour before she knew anything. When her mother subsequently asked, 'Why did you drop the child?' she said, 'I cannot remember anything about how it happened.' That four or five years ago she found herself on the roof of a house, when all at once she saw everything red and black before her eyes; that she was interrogated as to what had happened but did not remember anything except the feeling of dizziness and the flashes of light; that on this occasion she was observed to rush toward the edge of the roof as if she intended to throw herself down; that before she jumped she was seized and carried downstairs while in contortions. After she came to and was asked why she wanted to throw herself from the roof, she said she 'didn't remember anything about it.'

"Assume that the youngest sister of X was eleven years old; was a constant sufferer from headaches, which occurred about twice a week and were often accompanied by feelings of dizziness, during which she was obliged to hold on to something to prevent falling; that about two years ago while at home her head suddenly went around and she fell unconscious; that preceding this attack she had a headache. When she came to and found herself on a sofa, her mother asked her what was the trouble, and she replied that she had no recollection of what had happened after she had felt her head going around; that after this attack her clothes and the floor were wet from micturition.

"Assume that X, from the age of about from two months to seven years, had been afflicted with convulsions; that up to her fifth year she wet her bed; that when she was about four years of age, she was sitting on a stool and suddenly fell to the floor, rigid, shaking, and staring, with her mouth distorted and frothy; that she was picked up, afterward remaining three or four hours in a state of unconsciousness or sleep, and on awakening she was in a state of stupidity. Assume that at ten years of age she was found lying on the ground bleeding about the face, with her mouth distorted and unconscious; that at this time she cut her forehead, the scar from which still remains; that on this occasion she was asked how she had come to be upon the ground bleeding, and replied that she did not know; that when her head became dizzy, she saw something red like a flash of light, and that the next thing she remembered was when she found herself upon her bed and her mother asked her how she got on the floor; that after the flash of light appeared, she could see nothing, and everything became black before her eyes.

"That at the age of about nineteen, X came from Italy to America and began work at plain sewing; that when about twenty years of age she was talking one day with members of her family, when suddenly she uttered a scream, ran toward the window, and was in the act of throwing herself therefrom, when she was prevented by a member of the family; that she was then placed on a chair, and at once began to tremble

and shake all over, and finally to knock her head against the wall; that she then became relaxed and remained powerless until placed upon a bed, where she apparently slept for some hours. After awakening and on being asked why she had sought to throw herself from the window, she answered, 'You are crazy, I don't remember that I ever tried to throw myself from the window'; that prior to this act she had felt her head 'going around' and saw something in her eyes just as she lost consciousness. On a later occasion, she suddenly started to run to the roof of the house and was caught by her father in time to prevent her from jumping from the roof; that he took her down and placed her upon a chair, when she then began to shake, with her arms stiff and her head thrown backward; that she then seemed to fall, and awoke after a couple of hours or so, and when asked what the trouble was, replied that she did not remember anything. That just previous to this X saw 'red and black' and felt something 'awful hot' in her head.

"That about three years ago she met a man who became exceedingly persistent in his attentions to her. Up to that time she had had no intercourse of even a social nature with men outside the members of her own immediate family. On or about March 28, 1895, after a pursuit of several months, after repeated endeavors on the part of X to avoid him, this man, subsequently deceased, took her into a saloon and gave her some liquid of a reddish color to drink, which she could smell, and which made her immediately ill, and while in that condition she was carried by him into a room and there dishonored.

"Assume that between the time of the aforesaid seduction and April 26, 1895, with the exception of two or three days directly after the seduction, X lived with this man in his apartments; that during this time she was possessed of one paramount idea and purpose, namely, to become the wife of her seducer in order to be able to return to the home of her parents; that she continually implored and prayed this man to marry her and save her honor. That although often struck and beaten by him, she did not evince any anger or menace him in any way whatsoever; that her de-

meanor and deportment during this time were tearful, despairing, and beseeching; that she lost weight and declared she would take her life unless she could marry and return to her parents' home; that during this time she was frequently seen sitting with arms and hands folded, head down, unoccupied, when suddenly she would burst into a fit of weeping. Assume that in the hearing of X the deceased boasted of seductions he had made in cases other than that of X; that he showed to X pictures of other women he had ruined, whose dishonor was his boast.

"Assume that ten days before the death of her seducer she was heard to utter a scream and seen to fall; that she had twitchings and contractions of the muscles; her limbs and body grew stiff and she was placed upon a bed in a seemingly unconscious condition; that her body lay rigid like a corpse, her face white, with froth about the mouth, and that she remained in this condition for eight or ten minutes; that when she came to she seemed dazed and stupid, and on being questioned appeared not to know what had happened to her.

"Assume that on the morning of the 26th of April, X and the deceased had their usual discussion concerning marriage, wherein he reiterated his refusal to accede to her request; that after this discussion the deceased went to a saloon, two doors from the house in which he and X lived; that within a few minutes of his arrival there X entered the saloon, when she again asked him to marry her, and he refused; that she then left the saloon and went into the house where she lived to get a piece of cloth for personal use from her trunk; that she was seized with an impulse to destroy herself and that when her hand accidentally came in contact with a razor, she took it, not with any intent of using it upon the deceased, but to assist her in executing her impulse; that she did not remember where she put the razor, and, leaving the house with suicidal intent still in mind, started to cross the street, the saloon in which she had left the deceased being on the same side of the street as the house in which she lived; that she hesitated in the middle of the street and then returned in the direction of the saloon with the desire to see

her seducer again before she died; that when she got
back to the saloon, she found her mother there in
tearful conversation with her lover, beseeching him to
marry her daughter, X; that X then said, 'Don't cry,
mother, perhaps he will marry me'; that X was herself
in tears at this time; that the discussion as to marriage
continued between X, her mother, and the deceased;
that the demeanor of X during this conversation was
tearful, imploring, and sorrowful; that the discussion
was carried on very quietly, and during the course
of it the deceased asked the defendant's mother for
money, as a price for which to purchase his consent to
marry her daughter; that finally the deceased was
heard to refuse the request of mother and daughter
and say, 'Pigs may marry,' or words of like import;
that instantaneously with the uttering of these words
X approached the deceased and inflicted the wound
which caused his death; that up to the time of the
infliction of this wound X remained tearful and sor-
rowful in appearance, but at the moment these words
were said she shook all over, her eyes were injected
and rolling, her whole appearance changed; that she
was heard to emit some sound or screech immediately
before the fatal act. She moved away from him in the
direction of the door of the saloon and fell several paces
from where she had wounded him, no one being near
her when she fell; that the wound inflicted by her was
made by a razor, beginning about two inches to the
left of the median line of the neck, passing across the
neck backward up to the bony prominence behind the
right ear, severing all the tissues in the neck so as to
expose the spinal column. Directly subsequent to the
infliction of the fatal wound, she, in some way un-
known, arose and was next seen on the sidewalk in
front of the saloon, gesticulating and uttering words
of the following import: 'Die, you have taken my vir-
ginity.' That at this time she had froth about her
mouth, her face was white and her eyes like glass and
staring; that she was looking at her blood-stained
hands and rubbing them the one upon the other; that
she took up her skirt and removed the foam or froth
from her mouth and then put her hands into a pail
of water standing near; that when she heard the words,

'pigs may marry,' she felt something very hot in her head and saw flashes of light, black and red, and then became blind; the next sensation that she remembered was when she saw her hands covered with blood. That she had no recollection of having injured or wounded the deceased, and thought that she herself was wounded; that she felt something in her mouth like soap, which she wiped off with her dress.

"Assume that after dipping her hands into water as above referred to, she entered a grocery store and was there taken into custody by an officer; that before and after her arrest she was passive and indifferent, making no effort to escape or in any way interfere with the officers of the law. Assume that after being arrested she went with the policeman to the apartments in which she had lived, where she removed her waist and threw it out of the window; that while in this room she was quiet and passive, and on arrival at the station house she still seemed unconscious, as though nothing had happened.

"That while at the police court on the day of the homicide, she seemed dazed, bewildered, and irrational, sitting fifteen minutes in a chair almost motionless, with her eyes half closed, fixed and staring; that she was addressed through an interpreter during this time and it was only after repeated questions that an answer or an apparent answer could be elicited; that as soon as she would seem to recover from her condition of apparent stupor and respond to a query, she would again relapse into the condition above described; that on the day of the homicide or shortly thereafter, and for a period of about two and a half months she was confined in prison, awaiting trial, during which period her condition was almost uniformly that of one dazed and stupefied, except that at not infrequent intervals she would have fits of weeping, would refuse food to a marked degree and suffered from sleeplessness.

"Assume that she was tried for the murder of her seducer on July 8, 1895, and the days following; that during the progress of the trial she was nervous and otherwise perturbed to such a degree that the trial had to be stopped in at least three instances before she recovered sufficiently to have the trial proceed; that

she was immediately, after the conclusion of this trial, taken to prison, there to await the result of an appeal which had been taken from the judgment of conviction. Assume that during her ten months' stay in prison her life was marked by symptoms of confusion and dullness at first, accompanied by bursts of weeping and later by a more conscious and continuous sorrow and despair; and that in April of 1896, when told she was to have a new trial, she seemed totally uninterested and indifferent at the news. That she had no recollection of the killing of her seducer, and knew that he had been wounded and had died only through information received by her from others.

"Assuming the statements in that question to be true: Are you of the opinion, or are you not of the opinion, that on the 26th of April, 1895, at the time of the killing of the deceased by the defendant, that this defendant was able to discriminate between right and wrong, or to know the nature and quality of the act she was doing?

"*Answer:* I am of the opinion that she was not able to do that."

The same question was answered in the negative by a large number of prominent alienists called by the defense.

Murder by an epileptic (Clouston) should usually be looked upon as being as much a symptom of his disease as larceny by a general paretic.

Impulsiveness is the dominant feature of most of the serious crimes committed by epileptics. Motive, as well as premeditation, is generally lacking. In many homicidal acts committed by epileptics, consciousness is retained and memory may not be destroyed, at any rate not immediately, although subsequently all recollection of the deed is lost.

Amnesia should not be confused with unconsciousness in such cases. The two are not identical; unconsciousness being a cessation of all psychic activity, naturally includes amnesia, while the latter does not necessarily include the former.

The retention of consciousness (Baker) and even memory is, perhaps, most often seen in cases in which there exists a pre-epileptic condition of suspicious or smouldering hatred fanned into the highest intensity in the post-epileptic state. Consciousness and memory are not, therefore, inconsistent with epileptic furor.

In post-epileptic automatism, amnesia is usually complete, though in some cases there may be a glimmering of consciousness resulting in the memory of certain things here and there that occurred during such state. (This is illustrated in the case of A. T. mentioned in the preceding chapter.) When things are remembered in part, the amnesic state is not complete, and in such form it is usually accompanied by some degree of psychomotor violence. Generally the violence is intense and may be prolonged for hours. It has been my observation that complete amnesic states are more often characterized by comparative silence; at least there is no movement or commotion in them that could be regarded as pathologic.

Many epileptics suffer from post-convulsive automatism in a manner not recognizable by the unskilled observer. I have previously cited numerous instances of the kind. Crimes committed in such states are essentially beyond the knowledge of the individual who commits them.

Dr. Orange (Peterson and Haines) relates the case of a mother "who, while cutting bread for her family, having her baby on her arm, became momentarily unconscious. On return of consciousness she proceeded in an automatic way to use the knife, not upon the loaf but upon her child, whose arm she amputated."

Premeditated criminal acts by epileptics are not common, though I recall several cases in which the motive was flimsy and the degree of injury sought to be inflicted out of all proportion to the apparent sense of personal grievance the epileptic based his assault upon.

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A male epileptic at the Colony got out of bed too early one morning and was told by the nurse to go back. The patient objected and the nurse insisted. The nurse left the room for a few moments, and on returning found the patient sitting on the bed trying to conceal something under the .covering. When the nurse asked what it was, the patient sprang at him and viciously stabbed him with a knife. It could not be determined that the patient had suffered from a seizure just before the assault, but his radical and sudden change in conduct led to such a belief. He had full subsequent recollection of the event. If he did not have a seizure just before the assault, it was committed under the duress of epileptic frenzy, a state characterized by absolute loss of inhibition.

Another patient started into the dining-room with trouser guards about his ankles. He was requested by the attendant to remove them, but profanely refused. On being sent away from the room, he turned as though to obey, but suddenly drew a knife, whirled around and stabbed the nurse in the shoulder, repeating the blow as the nurse turned to face him.

An asylum physician in France going his rounds stopped to talk with a male epileptic employed as a tailor. The physician was stooping to examine the patient's leg, as he complained of pain in it. Suddenly the man passed his arm around the physician's neck, and thrust his long tailor's scissors into his side, causing death. The act was without motive. ("American Journal of Insanity," Vol. xiv, p. 309.)

A woman of twenty years, whose *grand mal* attacks all occurred at night, constantly manifested a most vicious disposition. On several occasions she assaulted nurses and fellow-patients weaker physically than herself, always choosing a time when her victim was alone. On one occasion, she threw an irritating washing-powder in the attendant's eyes, preliminary

to an attempt to throw her from a window. When accused, she tearfully protested her innocence. She was later committed as insane.

Article 64 of the French penal code contains this paragraph in reference to mental affections: "There can be neither crime nor offence when the accused was in a state of dementia at the time of the act, or when he was restrained by a force which he was powerless to resist" (Féré).

This is an admirable provision for the protection of the epileptic who unwittingly commits a crime while under the overpowering duress of his disease.

The French Society of Legal Medicine recommended in 1875 that the general rules governing the examination into the responsibility of the insane likewise apply to epilepsy. We cannot protest too earnestly against the legal inequality established in favor of the insane.

Epilepsy due to accident or injury may be a basis for legal claim for damages. Pearce Bailey * very lucidly discusses the features of this contingency in substance as follows:

It may be easy or exceedingly difficult to establish causal relationship between an injury and epilepsy. If there is evidence of a fracture of the skull, the matter will be simpler than when no evidence of injury exists. It must be shown that the patient never had convulsions prior to the receipt of the injury, except such as may have occurred in infancy and which are not regarded as an expression of idiopathic epilepsy.

Alcoholism and other degenerative conditions that may have existed immediately before the injury must be considered, especially if the injury is supposed to have been slight.

If the injury results in hemiplegia or monoplegia with convulsions following, the cause is plain; or if

* "Accident and Injury," 1898, p. 134.

the seizures are local and so indicative of localized brain lesion, there will rarely be any question as to their traumatic origin.

The time elapsing between the receipt of the injury and the first epileptic manifestations play little part in diagnosis. Competent statistics show a wide variation in this respect. In some the seizures appear immediately; in others, not for months or possibly not until years after degenerative changes have become definitely established.

In some cases age may assist in diagnosis. Idiopathic epilepsy is essentially a disease of early life.

Bailey remarks, in conclusion, that "no case of epilepsy in which there is any question of operation or of medicolegal inquiry should be accepted as traumatic, unless the seizure has been witnessed by a physician or by some person trained in the observation of the symptoms of the disease. Such observations can best be carried out in a hospital."

Epilepsy and Life Insurance.—The medical jurisprudence of life insurance in its application to epilepsy may seek to distinguish between "fits" and epileptic convulsions. In Hamilton's "System of Legal Medicine" we find this: "The term 'fits' is so vague that it has no standing medically. The present tendency is to construe it as referring to epileptic convulsions alone. In the old application, some importance was attached to it. As the distinction between the different varieties of spasms was not then understood, the term probably included other convulsions than epileptic."

One Griswold (Chattock *vs.* Shawe *et al.*, "3 Bigelow's Life and Acct. Ins. Rep.," 10) was insured in 1831, stating at the time that he was "in a sound and perfect state of health, and had not been afflicted with or was subject to . . . fits, etc."

It was proved that he had had two fits of an epileptic

character in 1827, but testimony was offered to show that they were the result of an injury to the head.

In his charge to the jury the judge said: "The interpretation I put on a claim of this kind is, not that the party never accidentally had a fit, but that he was not, at the time of the insurance being made, a person habitually or constitutionally afflicted with fits, or a person liable to fits from some peculiarity of temperament, either natural or contracted from some cause or other during life."

The extraordinary features of this charge become apparent when we recall the frequency of trauma as a cause of epilepsy.

Some life insurance companies issue standard policies to persons medically regarded as having under-average lives. Epileptics belong to this class. The average life of those having *grand mal* is shorter than those having *petit mal* attacks. The specific form of the disease fixes the rate of insurance premiums, which in any epileptic is higher than in non-epileptics. Other companies refuse to insure epileptics at all, and hold that epilepsy concealed from the company's knowledge, when the policy is issued, is sufficient to invalidate the same.

The testamentary capacity of epileptics may be questioned in relation to the execution of wills. Hamilton * cites the case of a male epileptic who made his will during the inter-paroxysmal period and was sustained. The testator, who was subject to attacks of acute mania due to epilepsy, was committed to a lunatic asylum on April 29, 1879, and discharged May 12, 1879. The superintendent of the asylum testified that the testator's mental condition was then good, and that during the interval between his attacks he possessed sufficient mental soundness to understand what he was doing.

* "System of Legal Medicine," Vol. II, p. 122.

The testator executed his will May 21, 1879. His family physician testified that he was of sound mind and memory and capable of making a will.

He was again attacked with an epileptic convulsion on June 11, 1879, and died June 24, 1879. It was held that the testator was sane at the time the will was made.

Hamilton (*op. cit.*) mentions another case in which a woman had an epileptic fit at the age of sixty-two, which rendered her unconscious. This was followed by pneumonia accompanied by high fever and delirium which also produced unconsciousness. She had previously been robust and intelligent.

Witnesses who were present during her illness stated that while occasionally out of her mind, she was at other times rational and intelligent, her mental condition being clearly the result of delirium attendant on high fever. No witness claimed that she had wholly lost her reason at that period.

She executed her will on November 23, 1868, and made three codicils between that time and her death, which occurred in July, 1875. Witnesses to the will and codicils testified to her sanity at the time she executed them.

When the case was tried in the Circuit Court of St. Clair County, in April, 1877, the jury found that she was insane at the time the will and codicils were executed, which rendered them null and void. This finding was reversed on appeal to the Supreme Court (Brown *vs*. Reggin, 94 Ill. R. 560).

The only way in which physicians can give testimony of value in such cases is for them to possess a thorough knowledge of the manner and extent to which epilepsy may affect intelligence temporarily or permanently.

Some epileptics are clearly competent during the inter-paroxysmal period to execute instruments whose purport is obvious to them at the moment. It does

not follow, however, in any case that the epileptic's action in such a matter may be remembered by him for any length of time afterward. A seizure occurring immediately after may efface the memory of it as completely as though it had never been received.

One of the most difficult questions to solve in respect to the testamentary capacity of epileptics pertains to the presence of amnesic states that come and go without any commotion whatever, and that may even escape the observation of those who may have the individual in view at the moment. These states may be prolonged for indefinite periods, during which the person acts in so nearly a normal manner as to be apparently under the guidance of full judgment and reason.

In addition to this periodic loss of conscious action, the majority of epileptics sooner or later attain some degree of mental impairment. To show in part the extent to which such impairment prevails, I present the results of a simple though effective test made at the Craig Colony in relation to the general intelligence of 801 epileptics.

TABLE SHOWING THE RESULTS OF TESTS OF THE GENERAL INTELLIGENCE IN 801 PATIENTS AT THE CRAIG COLONY, OCTOBER 1, 1903.

		MEN.			WOMEN.		
		Yes.	No.	Total.	Yes.	No.	Total.
Can the patient tell	name	441	22	463	319	19	338
	age	357	106	463	278	60	338
	year	312	151	463	222	116	338
	month	305	158	463	233	105	338
	day of week	331	132	463	244	94	338
	when born	343	120	463	220	118	338
	in what year born .	271	192	463	152	186	338
	name of last residence	363	100	463	255	83	338
	name of institution	348	115	463	234	104	338
	how long here	320	143	463	233	105	338
	sign name	331	132	463	246	92	338

The preceding table shows that out of 801 patients at the Colony on October 1st, 41 could not tell their names; 166, their age; 267, the year; 263, the month; 226, the day of the week; 238, when they were born; 378, the year of their birth; 183, their last place of residence; 219, the name of the institution they were in; 248, the length of time they had been there; while 224 could not write well enough to sign their names.

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